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TRANSACTIONS

OF THE

AMERICAN PEDIATRIC SOCIETY

SEVENTEENTH SESSION

HELD AT HOTEL SAGAMORE, LAKE GEORGE, N. Y.,
JUNE 19, 20 and 21, 1905

EDITED BY

LINNÆUS EDFORD LA FÉTRA, M.D.

VOLUME XVII.

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PRESIDENTS.

1889. A. Jacobi, M.D.
1890. J. Lewis Smith, M.D.
1891. T. M. Rotch, M.D.
1892. Wm. Osler, M.D.
1893. A. D. Blackader, M.D.
1894. John M. Keating, M.D.
1895. F. Forchheimer, M.D.
1896. Joseph O'Dwyer, M.D.
1897. Samuel S. Adams, M.D.
1898. L. Emmett Holt, M.D.
1899. Wm. P. Northrup, M.D.
1900. Henry Koplik, M.D.
1901. Wm. D. Booker, M.D.
1902. W. S. Christopher, M.D.
1903. J. P. Crozer Griffith, M.D.
1904. Augustus Caillé, M.D.
1905. C. G. Jennings, M.D.
1906. A. Jacobi, M.D.

OFFICERS, 1905.

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Recorder and Editor.....L. E. La Fétra, M.D.

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Floyd M. Crandall, M.D.
Thomas Morgan Rotch, M.D.
F. S. Churchill, M.D.
L. Emmett Holt, M.D.
John Lovett Morse, M.D.

MEETING PLACES.

1888. Washington, D. C. (Organization), September 18.
1889. Washington and Baltimore, September 20 and 21.
1890. New York, June 3 and 4.
1891. Washington, September 22 and 25.
1892. Boston, May 2, 3 and 4.
1893. West Point, N. Y., May 24, 25 and 26.
1894. Washington, May 29 and June 1.
1895. Virginia Hot Springs, May 27, 28 and 29.
1895. Montreal, May 25, 26 and 27.
1897. Washington, May 4, 5 and 6.
1898. Cincinnati, June 1, 2 and 3.
1899. Deer Park, June 27, 28 and 29.
1900. Washington, May 1, 2 and 3.
1901. Niagara Falls, May 27, 28 and 29.
1902. Boston, May 26, 27 and 28.
1903. Washington, May 12, 13 and 14.
1904. Detroit, May 30, 31 and June 1.
1905. Lake George, N. Y., June 19, 20 and 21.
1906. Atlantic City, N. J., May 30, 31 and June 1.

OFFICERS, 1906.

President.....A. JACOBI, M.D.
First Vice-President.....F. HUBER, M.D.
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Secretary.....SAMUEL S. ADAMS, M.D.
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F. S. CHURCHILL, M.D.
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JOHN LOVETT MORSE, M.D.
S. McC. HAMILL, M.D.

MEMBERS.

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ACKER, GEORGE N., M.D.....913, Sixteenth Street, Washington
ADAMS, SAMUEL S., M.D.....1, Dupont Circle, Washington
BAINES, ALLEN, M.D.....194, Simcoe Street, Toronto
BLACKADER, A. M., M.D.....236, Mountain Street, Montreal
BOOKER, WILLIAM D., M.D.....208, West Monument Street, Baltimore
BUCKINGHAM, E. M., M.D.....346, Marlborough Street, Boston
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EDSALL, D. L., M.D.....1432, Pine Street, Philadelphia
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GRIFFITH, J. P. CROZER, M.D.....1810, Spruce Street, Philadelphia
HAMILL, S. McC., M.D.....1822, Spruce Street, Philadelphia

HAND, ALFRED, JR., M.D.....1724, Pine Street, Philadelphia
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LADD, MAYNARD, M.D.....220, Marlborough Street, Boston
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MCCOLLOM, JOHN H., M.D.....745, Massachusetts Avenue, Boston
MARTIN, C. F., M.D.....33, Durocher Street, Montreal
MILLER, D. J. MILTON, M D.....1801, Pine Street, Philadelphia
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STARR, LOUIS, M.D.....1818, South Rittenhouse Square, Philadelphia
TOWNSEND, CHARLES W., M.D.....76, Marlborough Street, Boston
WENTWORTH, A. H., M.D.....352, Marlborough Street, Boston
WEST, J. PARK, M.D.....3608, Belmont Street, Bellaire, Ohio
WESTCOTT, THOMPSON S., M.D.....1833, Spruce Street, Philadelphia
WILLIAMS, HAROLD, M.D.....528, Beacon Street, Boston
WILSON, W. REYNOLDS, M.D.....1709, Spruce Street, Philadelphia
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YALE, LEROY M., M.D.....432, Madison Avenue, New York

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DR. JOHN THOMPSON.....Edinburgh, Scotland
DR. GEORGE F. STILL.....London, England
DR. HENRY ASHBY.....Manchester, England
DR. O. HEUBNER.....Berlin, Germany
DR. THEODOR ESCHERICH.....Vienna, Austria
DR. WILLIAM OSLER.....Oxford, England
DR. A. BAGINSKY.....Berlin, Germany
DR. V. HUTINEL.....Paris, France

Deceased.

JOHN A. JEFFRIES, M.D.
Born, September 2, 1859,
Died, March 26, 1892.

THOMAS F. SHERMAN, M.D.
Born, March 17, 1856,
Died, September 26, 1893.

JOHN M. KEATING, M.D.
Born, April 20, 1852,
Died, November 17, 1893.

CHARLES WARRINGTON EARLE, M.D.
Born, 1845,
Died, November 19, 1893.

J. LEWIS SMITH, M.D.
Born, October 15, 1827,
Died, June 9, 1897.

JOSEPH O'DWYER, M.D.
Born, October 12, 1841,
Died, January 7, 1898.

JOHN HENRY FRUITNIGHT, M.D.
Born, November 9, 1851,
Died, December 18, 1900.

FREDERICK A. PACKARD, M.D.
Born, November 17, 1862,
Died, November 1, 1902.

WALTER S. CHRISTOPHER.
Born, 1859,
Died, March 2, 1905.

Obituary.

WALTER S. CHRISTOPHER, M.D.

Dr. Walter S. Christopher, a collaborator of ARCHIVES OF PEDIATRICS, one of the best known and best loved physicians of this country, died on March 2, 1905, at his home in Chicago.

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His childhood was spent in Cincinnati, O., where he was educated, graduating from the Medical College of Ohio in 1881. Early in his professional career he was elected demonstrator of chemistry in his *alma mater*, and consulting chemist to the Rookwood Pottery. During his connection with this latter institution he perfected some of the glazes which have since given world-wide fame to the Rookwood ware.

Soon after his graduation he was made clinician to the Children's Clinic of the Medical College of Ohio, which identified him with that department of medicine, the study and teaching of which were to become his life's work.

In 1889 he was made professor of medicine in the University of Michigan. In 1890 he moved to Chicago and was elected professor of pediatrics in the College of Physicians and Surgeons of that city. He was elected a member of the American Pediatric Society in 1889, and was president of this society in 1902.

Dr. Christopher was an enthusiastic pediatrician and contributed to the literature of this branch of medicine many valuable papers. He was especially interested in the intricate and obscure chemical problems associated with nutrition during the early developmental periods and the many contributions from his pen on these subjects will remain as a lasting monument to his memory. Perhaps his most notable contributions are those derived from the "Child-Study" investigations made in the Chicago public schools. This work ranks with the best of its kind that has yet been attempted, and led to the establishment under his direct supervision of a department for "Child-Study" in the Chicago public schools.

During the last ten years of his life, Dr. Christopher devoted his whole time to the study, teaching and practice of pediatrics. He was recognized as the leading pediatrist of the Northwest. His untimely death at the age of forty-six was largely due to his unselfish devotion to his friends and to his profession, which he loved better than he did himself.

CAT.

WALTER S. CHRISTOPHER, M.D.

MINUTES OF THE SEVENTEENTH ANNUAL MEETING OF THE AMERICAN PEDIATRIC SOCIETY.

Held at Hotel Sagamore, Lake George, N. Y., on June 19, 20 *and* 21, 1905.

The following members were present: Doctors I. A. Abt, Chicago; G. N. Acker, Washington; S. S. Adams, Washington; A. D. Blackader, Montreal; W. D. Booker, Baltimore; E. M. Buckingham, Boston; W. L. Carr, New York; H. D. Chapin, New York; F. M. Crandall, New York; D. L. Edsall, Philadelphia; E. E. Graham, Philadelphia; J. P. Crozer Griffith, Philadelphia; A. Hand, Jr., Philadelphia; L. E. Holt, New York; F. Huber, New York; A. Jacobi, New York; C. G. Jennings, Detroit; C. G. Kerley, New York; H. Koplik, New York; L. E. La Fétra, New York; J. H. McCollom, Boston; C. F. Martin, Montreal; J. L. Morse, Boston; T. M. Rotch, Boston; E. W. Saunders, St. Louis; H. L. K. Shaw, Albany; I. M. Snow, Buffalo; C. W. Townsend, Boston; A. H. Wentworth, Boston.

The following visitors were present:—Doctors C. F. Gardner, Colorado Springs; A. R. Mills, University of Sydney, Australia; G. R. Pisek, University of Vermont.

FIRST SESSION, JUNE 19, 2 P.M., HOTEL SAGAMORE.

Dr. J. P. Crozer Griffith, of Philadelphia, read a paper on "Sclerema Neonatorum."

Discussion by Drs. Koplik, Jennings, Jacobi and Griffith.

Dr. J. Lovett Morse, of Boston, presented a paper entitled, "Acid Autointoxication in Infancy and Childhood."

Discussion by Drs. Edsall, Koplik, Holt, Rotch, Crandall, Townsend, Jacobi and Morse.

Dr. Henry Dwight Chapin presented "Notes on the Examination of the Urine of Infants: (*a*) In Digestive Disturbances; (*b*) In Pulmonary Disturbances; (*c*) In Other Conditions. Methods of Collecting Urine for Examination."

Discussion by Drs. Holt, Edsall, Crandall, Morse, McCollom, Jacobi, Jennings, Koplik and Chapin.

Dr. A. Jacobi, of New York, reported "A Case of Sepsis in the Newlv-born."

Discussion by Drs. Holt, Huber, Koplik, Edsall, Saunders and Jacobi.

Dr. A. H. Wentworth, of Boston, reported "A Case of Acute Yellow Atrophy of the Liver in a Child."

Discussion by Dr. Griffith.

Dr. L. Emmett Holt, of New York, read a paper on "Multiple Tumors of the Abdomen."

Discussion by Drs. Blackader, Buckingham, Huber and Holt.

Dr. Charles G. Jennings, of Detroit, reported "A Case of Abdominal Tumor."

SECOND SESSION, JUNE 19, 8 P.M., HOTEL SAGAMORE.

Dr. J. P. Crozer Griffith presented the "Report of a Case of Stenosis of the Pylorus in Infancy."

Drs. Thomas Morgan Rotch and Maynard Ladd, of Boston, reported "Two Operative Cases of Pyloric Stenosis in Infants."

Discussion of the two papers by Drs. Morse, Abt, Holt, Griffith and Rotch.

Dr. E. W. Saunders, of St. Louis, read a paper entitled, "A Plea for the More Timely Use of Intubation in Laryngeal Stenosis, and for the Use of Tracheotomy in Certain Neglected Cases."

Discussion by Drs. Chapin, Booker, McCollom, Adams, Crandall, Abt and Saunders.

Dr. George N. Acker, of Washington, reported "A Case of Porencephalus."

Discussion by Dr. Abt.

Dr. Henry Koplik, of New York, presented "A Contribution to the Pathology and Causation of Congenital Stridor: Case and Autopsy."

Discussion by Drs. Morse, Griffith, Blackader, McCollom, La Fétra and Koplik.

Dr. Henry L. K. Shaw, of Albany, read a paper on "The Use of Citrate of Soda in Infant Feeding."

Discussion by Drs. Graham, Chapin, Abt, Holt, La Fétra, Kerley and Shaw.

THIRD SESSION, JUNE 20, 10 A.M., AT THE RESIDENCE OF DR. JACOBI, BOLTON LANDING.

The annual address of the president, entitled "The Medical Supervision of Schools and the Progress of School Hygiene," was read by Dr. Charles G. Jennings, of Detroit.

On motion of Dr. Blackader the address was referred to the Council for consideration and report.

Drs. A. D. Blackader and A. D. Gillies, of Montreal, presented the report of "A Case of Acute Lymphatic Leukemia in a Child Aged Fourteen Years."

Dr. Alfred Hand, Jr., of Philadelphia, reported "A Case of Acute Lymphatic Leukemia in a Child Aged Five Years."

Discussion of both papers by Drs. Holt, Koplik, Edsall, Morse, Abt, Blackader and Hand.

Dr. Henry Koplik presented, for Dr. Henry Heiman, of New York, "A Study of the Leukocyte Count in the Bronchopneumonia, Lobar Pneumonia, and Empyema of Infants and Children."

Dr. Francis Huber, of New York, read a paper entitled, "Costal Synostosis about Drainage Tubes in Empyema."

Discussion by Drs. Graham, Koplik, Jacobi, Blackader, Kerley and Huber.

Dr. S. S. Adams reported "A Case of Uncinariasis in a Child."

Discussion by Drs. Acker, Graham, Rotch and Adams.

Dr. Charles W. Townsend, of Boston, reported "An Unusual Case of Tuberculosis in an Infant."

Discussion by Drs. Rotch, Blackader, Buckingham, Morse, Edsall, Saunders, Kerley, Blackader, Holt and Townsend.

Dr. Henry L. K. Shaw presented a case of "Congenital Heart Disease without Cyanosis."

EXECUTIVE SESSION, JUNE 20, 8 P.M., HOTEL SAGAMORE.

Dr. J. P. Crozer Griffith presented the report of the Council, upon whose recommendation the following action was taken:

The officers elected for the ensuing year were: President, Dr. A. Jacobi; First Vice-President, Dr. F. Huber; Second Vice-President, Dr. A. H. Wentworth; Secretary, Dr. Samuel S. Adams; Treasurer, Dr. J. Park West; Recorder and Editor, Dr. Linnæus E. La Fétra.

Dr. Samuel McC. Hamill was elected a member of the Council to succeed Dr. F. Forchheimer, retired.

The Society voted to hold the next annual meeting at Atlantic City, N. J., May 30, 31, and June 1, 1906.

Dr. William Osler, Oxford, Dr. A. Baginsky, Berlin, and Dr. V. Hutinel, Paris, were elected to honorary membership.

Elected to membership were Dr. John Howland, New York, Dr. T. S. Southworth, New York, Dr. John Ruhräh, Baltimore, and Dr. J. H. Mason Knox, Baltimore.

The proposition of E. B. Treat & Co. in regard to the publication of the Transactions of the Society was accepted.

An assessment of $15 per member was ordered. To defray the expenses of the American Committee on the International Congress $25 were appropriated as the Society's proportion.

The treasurer's report was audited and found correct.

Upon nomination by the Council Drs. Jennings, Jacobi and Abt were elected members of the "Committee on the Welfare of Children." The objects of this committee are to look after the general and hygienic interests of children and especially to represent the Society in conferences with committees of educational and legislative bodies.

Dr. Jacobi was elected delegate to the Fifteenth International Medical Congress at Lisbon, 1906.

FOURTH SESSION, JUNE 20, 9 P.M., HOTEL SAGAMORE.

Dr. J. P. Crozer Griffith presented for Dr. Myer Solis-Cohen, of Philadelphia, a paper on "Temperature, Pulse and Respiration Relationships in Infancy and Childhood."

Discussion by Drs. Holt, Jacobi, Hand and Griffith.

Dr. D. L. Edsall, of Philadelphia, read a paper entitled, "Milky, Non-fatty Effusion in a Case of Hodgkin's Disease."

Discussion by Drs. Graham, Jacobi, Huber and Townsend.

Dr. E. E. Graham, of Philadelphia, presented a paper on "The Results in Children of Descapsulation of the Kidneys for Nephritis, with the Report of an Apparently Successful Case in a Child Aged Twenty-six Months."

Discussion by Drs. Huber, Pisek (guest) and Graham.

Drs. Alfred Hand, Jr., and J. C. Gittings, of Philadelphia, read "An Analysis of 145 Cases of Typhoid Fever in Children."

Discussion by Drs. Buckingham, Adams, Griffith, Blackader, Abt, Saunders and Hand.

The following papers were read by title:

Dr. W. P. Northrup, of New York, "High Per Cent. Fat in a Mother's Milk, Producing Severe Enterocolitis in Her Infant."

Dr. A. C. Cotton, of Chicago, "A Case of Congenital Exstrophy, with Extroversion of the Bladder and other Anomalies."

Dr. Irving M. Snow, of Buffalo, "The Eye Symptoms of Infantile Scurvy: Autopsy; Large Infected Retrobulbar Hematoma."

LINNÆUS E. LA FÉTRA, M.D.,
Recorder.

THE MEDICAL SUPERVISION OF SCHOOLS AND THE PROGRESS OF SCHOOL HYGIENE.

PRESIDENTIAL ADDRESS.

BY CHARLES GODWIN JENNINGS, M.D.,
Detroit, Mich.

Since we last met Christopher has departed from our councils. As a physician, as member, councilor and president of this Society, his geniality, intense humanity, and sturdy, broadly cultured mind placed him in the front ranks of the pediatrists of this country. His early end has left a place in our professional lives that it is not easy to fill, and we cannot but pause to express our profound sorrow and regret.

In his presidential address last year, my distinguished predecessor, Dr. Caillé, called the attention of the Society to the necessity of broadening the scope of its labors and of taking cognizance of public questions that relate to the hygiene and pathology of childhood. While, as shown by its proceedings, the activities of this Society have been rather closely confined to the investigation of the problems of the pathology and therapeutics of early life as they relate to the individual, its position as the representative association of this continent for the study of pediatrics demands that it should speak with its authority on the broader subjects connected with the hygiene of child life, on which the physical well-being of the race, and even our existence as a nation, depend.

Following the suggestions of Dr. Caillé the president addressed to each member of the Council, whose duty it is to formulate the work and policy of the Society, a communication asking for suggestions as to the advisability of an effort to increase the power and usefulness of the Society, and asking for topics for discussion, etc. While some were encouraging, the general tone of the replies was such as to give the impression to the president that the Council was satisfied with the work of the Society in the past and that no radical changes were desirable. I still believe, however, that Dr. Caillé's conception of the field of usefulness of the Society was right, and that future administrations should direct its efforts to the fulfilment of it.

Among other questions considered in his address, Dr. Caillé

adverted briefly to the subject of school hygiene and school inspection.

As one of the commissioners of the Department of Health in the city of Detroit, my duties have compelled me to keep informed upon the development and progress of medical inspection of school children. I believe the importance of the subject justifies its further and fuller consideration by the Society, and I, therefore, ask your attention today to:

THE MEDICAL SUPERVISION OF SCHOOLS AND THE PROGRESS OF SCHOOL HYGIENE.

Although Herbert Spencer and some of the ablest thinkers of our time have contested the right of the State to impose its system of culture upon the citizen, it has become a political axiom in the United States and many of the states of Europe that the government is responsible for the education of its citizens. Here and in Europe the common school has become a fact in the development of state socialism. Compulsory education is a settled state policy in the United States.

Forgetful of the importance of character formation, and still more forgetful of physical development in childhood, the attention of the State and of educators has been concentrated upon the small part of child education, the development of the intellectual capacity.

The compulsory removal of all children from the influence of home life and their segregation in school rooms for from five to six hours a day to receive the intellectual training the State demands, imposes upon the State the secondary, but not less important, duty of a medical supervision that shall indicate their mental and physical ability to endure intellectual training, guard their physical and mental well being during school life, and discharge them physically and morally, as well as intellectually educated, and ready to take up their life work as the fathers and mothers of the next generation.

Medical officers of health and physicians in private work meeting as they do the disasters to child life that come from enforced exposure in schools to infectious disease, from the unsanitary environment of crowded schoolrooms, and from the increased stress of life involved in the concentration of nervous effort at school work, are agreed that the time has come when children during school life should be protected from these dangers by adequate medical supervision. Before this Society of physicians, familiar

with the etiology of acute and chronic diseases of childhood, it is unnecessary to mention in detail the pathologic conditions directly and indirectly traceable to school life. It is equally unnecessary to emphasize the fact that the medical supervision of school children can be done only by medical men. The detection of the acute and chronic infections, of visual, auditory and other physical imperfections, of mental deficiencies and perversions, call for systematic inspection by skilled physicians.

In this address I wish briefly to bring before you the work that has been accomplished in the interests of a safer and healthier school life, the trend of the work of the present time, and the possibilities of the future.

Naturally, the most conspicuous danger of school life, exposure to the acute infections, was the first object of school inspection. With the attention of educators and parents, health officers and physicians directed to the subject, the necessity of amplification to a complete system of medical supervision of schools was gradually appreciated.

Medical inspection of schools had its beginning in Europe. As early as 1832 in Sweden, State regulation of school work was begun. The first laws relating to school physicians and school inspection were passed in France in 1833. The present system of school inspection in Paris was adopted in 1884. Impelled by the increasing myopia in Germany, Cohn, in 1864, inaugurated school inspection by the examination of the eyes of over 10,000 school children. Russia has appointed a physician to each secondary school since 1871. Belgium followed in 1874. In some cities or districts of most of the states of Europe, in Japan and in Egypt, medical inspection of some character has been begun.

Dr. Paul Schubert, of Nuremberg, in a recent monograph, "Das Schularzt wesen in Deutschland," has treated the subject of the school physician in Germany in a most exhaustive manner, and clearly sets forth excellencies and defects of the German system.

With characteristic seriousness and fully realizing the international importance of the subject, the German Union for School Hygiene, under the presidency of Professor Dr. Griesbach, called the First International Congress of School Hygiene, which convened at Nuremberg, April 4 to 9, 1904. The Congress met under royal patronage, and with the support of the various societies of Europe directly interested in the subject. The United States and Canada were represented by an organization of a president and

twenty-six members, several of whom were present in person. The transactions are published in four large volumes and is a remarkable series of papers and discussions, covering the whole field of school hygiene. Upon invitation of Sir Lauder Brunton, representing the Royal Sanitary Institute of Great Britain, the Second Congress will be held in London in August, 1907. The influence of this Congress has been of inestimable value in collecting and rapidly disseminating the facts and theories of school hygiene.

"In England the system has so far spread that already there is a Society of Medical Officers for Schools; the larger school boards provide for medical attendance and their teachers and pupil teachers, for the regular visiting of their schools, for the dovetailing of medical school inspections with the regular work of the public health organizations, and, generally, for the discovery of infection among school children and the prevention of infection by detailed periodic examinations of school premises. In many public schools, more especially where a boarding system is in force, medical inspection has gone much further. Such schools retain the services of a medical officer, whose duty it is to examine all pupils admitted, to ascertain their fitness both for the mental work of the school and for the physical training required, to attend any cases of illness that may arise, to arrange for the isolation of infectious diseases either within premises provided by the school or in the hospitals of the local authorities, and to report, from time to time, to the governors, any circumstances that may imperil the hygienic safety of the children. In the industrial schools, too, which are under the control of the Home Office, the managers appoint a medical officer whose duty it is to attend any cases of sickness, and to exercise a general supervision over the health of the school children. In many other schools, even where medical practitioners are not retained, it has become a custom systematically to measure, weigh, and examine all the pupils admitted, and, from time to time, all pupils in attendance." (Mackenzie.)

The works of Carpenter and Arthur Newsholme, the textbook of Mackenzie, the writings of Kerr, Clement Dukes, Chisholm, Lauder Brunton, and numerous physicians, educators and philanthropists, and, above all, the Conference on School Hygiene, organized by the Royal Institute of Hygiene in February of this year, reveal the fact that the people of Great Britain are fully alive to the national importance of this question.

Medical inspection of schools in the United States has been

slow of adoption and limited in its application. The diffusion of authority in municipal government, the occasional unfortunate introduction of partisan politics into educational administration, the democratic reluctance to the endurance of paternal measures of restraint, all have had their influence to delay the general adoption of medical supervision of school children. Again, only in the last few years has the density of urban population increased to the extent that the necessity of medical control of the aggregations of children in the common schools became apparent.

Boston introduced the first system of medical inspection of schools in 1894. New York followed in 1897. To assure themselves of the value and necessity of a system of inspection, the boards of health of New York and Boston conducted a series of preliminary investigations. To the members of this Society familiar with the modes of transmission of the infectious diseases, it is unnecessary to detail these investigations. They were made to assure administrative and legislative bodies and to demonstrate to them facts of common medical knowledge.

Chicago, Philadelphia, St. Louis, Washington, Detroit, and many of the smaller cities of the United States, have followed the lead of Boston and have adopted systems of medical school inspection.

With some exceptions, medical inspections of schools in the United States has had for its object only the detection and exclusion of the infectious diseases. It has been a measure originated and carried on by boards of health for the prevention of the spread of the contagious diseases of childhood, and until boards of education have adjusted themselves to this new duty, it will remain a department of health administration.

In all probability the medical inspection of schools should be under the control of the Board of Education. It should be a department of the school system and only related to the Board of Health as the two bodies may be of mutual benefit in performing their respective functions.

Wherever in the United States medical school inspection has been established, health officers have been quickly and fully convinced of its value in protecting the health and lives of children and in diffusing a knowledge of the principles and practice of preventive medicine. The value to a community of a system that has for its object only the exclusion of pupils suffering from contagious diseases is well illustrated by the records of the Depart-

ment of Health of Detroit. From the beginning of the school year in September, 1904, to June 1, 1905, there were examined by the medical inspectors 24,096 pupils. Of these, 1,678 were found to be suffering from the various communicable diseases, as follows:

Scarlet fever	17
Diphtheria	21
Measles	4
Mumps	28
Varicella	91
Rötheln	1
Pertussis	4
Tonsilitis	570
Contagious skin diseases and miscellaneous diseases	942

Reports from other cities show similar results. Regardless of such evidence that should be convincing, and like all measures for the protection of the community at the inconvenience of the individual, the medical inspection of schools has met with abundant opposition, and sometimes from unexpected quarters. A very recent editorial in a conservative and influential daily paper of Detroit well illustrates the need of public education in the value of preventive medicine. Speaking of the medical inspection of schools, this writer said: "It cost the taxpayer last year $2,500 for this perfectly useless service, a service that would be ridiculous were it not an abominable and intolerable piece of impertinence." It is almost pathetic to contemplate the state of mind of one who can designate as impertinent the protection by a department of health of hundreds of children from exposure to dangerous contagious diseases. It is gratifying to note, however, that with only an occasional exception medical school inspection has been received with approbation by boards of education, teachers and intelligent citizens.

Up to the present, the medical supervision of schools has not been considered seriously in the United States by national bodies. The United States Bureau of Education has published in the reports of 1898 and 1902 chapters reviewing the progress of medical inspection of schools in this country and abroad. In educational bodies, desultory discussions have arisen from time to time, and in 1898, at the meeting of the Department of Superintendence

at Chattanooga, a Committee on School Hygiene was appointed, with the United States Commissioner of Education, W. T. Harris, as chairman. This committee made a preliminary report the following year, giving the deplorable conditions existing in the public schools of this country and calling for the enlightenment of school boards, teachers and others regarding the subjects of school hygiene and sanitation. Although the report of this committee revealed the urgent need for an organized movement for the improvement of school sanitation and hygiene, no action was taken on the recommendation included in it.

The Section of Nervous and Mental Diseases of the American Medical Association, at the session of 1903, appointed a committee to inquire into the relation of school methods to school diseases. The members of the committee, Dr. W. J. Herdman and Dr. James H. McBride, reported to the Section at the session of 1904, and their report is published in the *Journal of the American Medical Association,* April 15, 1905. The report embodies a brief review of the progress of medical inspection, with comments, and this conclusion:

"In the opinion of your committee, the American Medical Association should place itself on record as advocating and urging the inauguration of a thorough and systematic medical inspection of the public schools and school children in every section of this country:

"1. In the interests of public health, since it is a potent means for detecting and preventing the spread of contagious and infectious diseases.

"2. For the purpose of securing to the child, while in attendance on school, the most favorable hygienic and sanitary conditions.

"3. For the purpose of getting exact knowledge regarding the physical and mental capacities of each child, in order that the methods of instruction may be intelligently directed to meet the individual needs.

"Thus far, it would seem we can safely go in support of what the most enlightened and progressive educators are seeking to accomplish, and in this effort they should receive from this national organization hearty endorsement and assistance, as they have in many localities from its individual members."

The American Academy of Medicine has appointed a committee to investigate the teaching of hygiene in the public schools.

Papers have been presented and the Academy is earnestly endeavoring to advance the progress of a reform movement.

Thus, it will be seen that the beginnings of a medical supervision of school life have brought to medical men and others a realization of its importance in the physical development of the race. Although at the present time efforts are confined almost exclusively to the discovery and exclusion of infectious diseases, a start has been made toward the development of a complete system of physical inspection. Chicago is studying the physiology and pathology of the school child. New York is beginning an investigation into the physical and mental condition of school children that is revealing a state of physical imperfection that would be startling were physicians not already too familiar with it. Of 7,166 boys recently examined by the medical inspector, 3,132 were found in need of medical attention; 650 of these showed defective mental development. The future will undoubtedly see the elaboration of medical school supervision until the whole period of school life is subject to a watchful care that will ensure education in its highest sense—the development of the entire mental, moral and physical faculties of childhood and youth.

PHYSICAL EDUCATION.—"The universal essential of growth is exercise. A harmonious training according to the entire nature is the condition of effective manhood. The development of mind is largely dependent upon efficiency of body; and the expansion of the moral element, so well expressed by the word character, is similarly intertwined: neglect of part is derangement of the whole.

"The character of a nation, as of an individual, is mainly dependent upon the completeness of the mental and moral education of its young; while the vigor and enterprise of a nation are based upon their physical training during the period of growth. No education is of permanent value which sacrifices health at its altar.

"The school motto must ever be, 'The greatest health of the greatest number.' The most sensitive test of a system, a school, or even a neighborhood, is the health, disease, and death-rate of the young whom it nurtures. To enable them to attain a bare existence is a grave neglect of social duty; to ensure their thriving is our bounden obligation. It is mainly during the years of school life, when its material is plastic, that a sound constitution can be produced, and a tendency to hereditary disease eradicated." (Dukes.)

In Great Britain, the startling announcement of the committee on the physical deterioration of the nation has been promptly followed by extensive inquiries into the physical conditions of school children in London, Edinburgh, Aberdeen, Glasgow and Dundee. With its less crowded population, this question has not yet become an urgent one in the United States, but the rapidly increasing urban population has brought emphatically to the minds of physicians that the time is soon coming when the questions of physical deterioration will here press for consideration as it now does in Europe.

From the conclusions of Clement Dukes in a paper read before the First International Congress of School Hygiene on "The Organization of Physical Education in Schools," I take the liberty of quoting: "The exercise of the physical faculties of children is as important for their well-being as mental and moral education. The development of these faculties is possible only during childhood."

"The physique of the next generation depends upon the physical education of the present, and physical education requires a similar organization to that of mental education.

"While the question of physical training is important enough in the case of boys, it is of equal or greater importance in the education of girls. In boys, the demand for exercise is spontaneous and irrepressible. Custom and false conceptions of physical grace hamper the whole childhood and youth of our women."

The serious defect in schools for girls is the inadequate provision for physical education. Women should be strong, vigorous and healthy, yet no effort is made during the period of their active growth and development to make them so. Education of growing girls should not be at the expense of motherhood; we do not want crammed heads on undeveloped bodies.

"If girls are to receive a higher culture, it is their physical education which must precede any increase in their mental education. Without this the process cannot be safely effected; for the mental powers, too highly developed in women, involve a physiological cost which her feminine organization will not sustain without injury more or less profound."

WORK AND SLEEP.—Knowledge of the physiology of childhood emphatically reveals how seriously inadequate is the provision for brain rest in the whole scheme of school education.

No attempt is made to adjust work and sleep to the physiological demands of the young, growing child. The work demanded of children in all grades is excessive, and this with the coincident deprivation of exercise, play, rest and sleep, is accountable for the illy-defined pathological conditions which medical men continually meet in childhood.

Clement Dukes has tabulated the hours of work and sleep requisite during youth and childhood, which is, in part, as follows:

THE HOURS OF WORK AND SLEEP ADAPTED TO THE VARIOUS AGES OF CHILDREN.

Ages of Pupils.	Hours of Work per Day.	Hours of Sleep per Night.
From 5 to 6 years.....	1	13½
" 6 " 7 "	1½	13
" 7 " 8 "	2	12½
" 8 " 9 "	2½	12
" 9 " 10 "	3	11½
" 10 " 12 "	4	11
" 12 " 14 "	5	10½
" 14 " 16 "	6	10
" 16 " 18 "	7	9½
" 18 " 19 "	8	9

As examples of the excessive work demanded in the school life of to-day, I will cite 3 cases recently seen, all suffering seriously from the physical effects of brain fag.

Case 1.—A daughter of wealthy parents in a private school for girls. School hours, 8:30 o'clock to 12:30 o'clock. Continuous study or recitation, with ten minutes' recess at 11 o'clock. Luncheon, 12:45 o'clock. German lesson and study, 1:15 o'clock to 2:30 o'clock. Play, from 2:30 o'clock to 4 o'clock. French or music, 4 o'clock to 5 o'clock. A total of six hours per day of school work.

Case 2.—A daughter of wealthy parents in a private school. The girl rose at 6 o'clock. Some hurried study, with breakfast at 7 o'clock. At school, from 8:30 o'clock to 12:30 o'clock, with fifteen minutes' recess at 11 o'clock. Luncheon at 1 o'clock. Music and recitation, 1:30 o'clock to 3:30 o'clock. Out until 6 o'clock. Dinner, 6:30 o'clock. Study, 7 o'clock to 10 o'clock. A total of

nine hours of work, one and one-half hours out of doors and eight hours of sleep.

Case 3.—A girl of seventeen years in the high school studying for a teacher's position. The girl rose at 6:30 o'clock. Breakfast at 7:30 o'clock, followed by hurried study. In school, from 8:30 o'clock to 1:15 o'clock, with fifteen minutes' recess for lunch. An indigestible lunch was often taken at recess. Dinner at 2 o'clock. Study, from 3 o'clock to 6 o'clock. Supper at 6 o'clock. Study, from 7 o'clock to 11 or 12 o'clock. A total of ten hours' study, six to seven hours' sleep and no recreation.

FOOD.—Ignorance of the simplest facts of dietetics in homes and in boarding-schools is responsible for many of the pathologic conditions of childhood. In addition to excessive work and long confinement, with deficient rest and sleep, is a dietary improperly selected, badly prepared and often inadequate to the demands of the growing child for nutrition.

These are but suggestions of the problems that demand correction in school life. How correction is to be made is a question for the earnest thought and united efforts of physicians, educators and parents. There are difficulties to be overcome that now seem insurmountable, but which with an aroused public sentiment may fade away. With a thorough medical supervision of the school system by a medical department of the Board of Education, with the school developed into a laboratory, where teaching of hygiene is made practical by a perfect sanitary environment, will come a diffusion of a knowledge of the necessities of healthful living that will make reform a natural process of development.

A broad-minded educator (Henderson, "Education and the Larger Life"), writing of the difficulties to be overcome in making over our primary schools, says:

"But the major difficulty, the one which may not in kindness be ignored, the one which prevents the humanizing of the lower schools, and vetoes many a wholesome, red-blooded experiment in education, is really this—when these children of good fortune, for such I must regard them, come, at fifteen years of age, to the door of the high school, they find it closed. They are not wanted. They do not know parsing and grammar and spelling and arithmetic and political geography and physical geography and history and civil government and physiology. They are simply strong and well, clear-eyed and accomplished, inquisitive and earnest, full

of power and promise. Comparing the two groups of utilities, the high school chooses the former. But often, it chooses with a sigh. What, then, is the excuse? It is the same excuse all along the line. The lower schools would be good if the high schools would let them, and the high schools would be good if the colleges would let them, and the colleges would teach the knowledge of most worth if the community would let them. Apparently, it is a superior madness which drives us."

It may not be the province of this Society to introduce reforms in school hygiene or other departments of national sanitation. The objects and methods of its organization preclude this. It may and should be ready, however, with its exceptional knowledge of child pathology, to unite with national educational bodies, boards of health, and similar public organizations, in movements directed to the improvement in the physical development and well-being of the children of the nation. To this end a standing committee could be appointed with power to represent the Society in national conferences and international congresses for the study of school hygiene. Such a committee would be the medium through which the special medical knowledge of the Society could be made available in the guidance of reform movements, and through it the Society kept informed of the development and progress of school hygiene.

Until the American Pediatric Society thus gives its aid to such problems of national development, it will have but partially fulfilled its duty to scientific medicine, and the welfare of the children of the nation.

CONGENITAL LARYNGEAL STRIDOR—A CONTRIBUTION TO THE PATHOLOGY OF THE AFFECTION WITH REPORT OF AN AUTOPSY ON A CASE.

BY HENRY KOPLIK, M.D.,
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Congenital laryngeal stridor is a peculiar symptom complex which has long been a matter of investigation and speculation. The symptoms are admitted by all to be connected with the larynx and not allied to other conditions simulating it in which the structures of the nasopharynx are involved. Laryngeal stridor is a peculiarity of respiration which appears or is noticed shortly after birth. The infant otherwise may be in apparent health; as the infant lies quietly in its crib or sits in the arms of the nurse a peculiar crowing or grunting sound is heard with inspiration. This sound may be scarcely audible, or it may be heard at some distance, as across the room and of a loud sawing timbre. As a rule, the sound is heard only with inspiration, but may in severe cases be present during expiration, also. At times in the mild cases the grunting sound becomes inaudible, but reappears at intervals. These infants, if they are caused to cry or become excited, may at the end of inspiration develop a piping noise similar to what is heard at the termination of an attack in laryngismus stridulus.

Though many of these infants are rachitic, because some of them are bottle-fed, they are not all so, for I have seen infants breast-fed and free from all signs of rachitis who presented the above symptoms complex. Nor do these infants necessarily present the Chvostek sign or any of the marks of "latent tetany," socalled, nor do all of them have socalled apneic attacks, although recently a case came under my observation—a newborn baby—which presented laryngeal stridor and apneic attacks. The picture, therefore, is a definite one, not necessarily merging with socalled convulsive respiratory conditions, such as laryngismus or apnea, common to infancy and childhood. Laryngeal stridor must not be confused with another condition which is

commonly met with in the newborn and in older infants and children. In this state there is noisy breathing, more or less marked, which dates from birth, but which is distinctly due to an adenoid obstruction in the nasal passages. These infants make a snoring noise, either when nursing or in undisturbed respiration, but the noise is distinctly posterior nasal in its origin. In these cases, as in laryngeal stridor, there may be a retraction of the lower part of the sides of the thorax and of the suprasternal notch in inspiration. These infants are distinctly lymphatic, but the cases of laryngeal stridor are not necessarily so.

In laryngeal stridor there may be inspiratory retraction of the suprasternal notch, though this is not always the case. The most marked cases I have seen had no inspiratory retraction.

The affection appears soon after birth; hence the term "congenital"; and may extend well into the period of childhood. Much has been written upon this affection, and confusion has been caused by the introduction of the term "thymic asthma" into its nomenclature. If the literature upon "thymic asthma" is sifted it will become apparent that all varieties of spasmodic respiratory conditions of infancy and childhood have at one time or another been grouped under this term. Laryngismus and apnea have been grouped as "thymic asthma." In other words, "thymic asthma" has been utilized to include spasmodic or convulsive respiratory conditions, in which the thymus was supposed, on account of its enlarged or distended condition, to exert some disturbing influence. The thymus has also been held to be a casual factor in the production of laryngeal stridor. It would be well were the term "thymic asthma" dropped, inasmuch as in all the conditions with which the thymus has been linked in the nomenclature of thymic asthma there is scarcely any socalled asthmatic condition present, but rather something outside of the lung proper as an *etiological* factor.

The *symptoms,* also, as far as the physical signs are concerned, do not draw the lungs into the picture, affecting only secondarily the rhythm of the respiratory act.

This brings us to a consideration of the various theories which have been elaborated to account for the production of "laryngeal stridor." The first theory is, that the symptom complex is caused by a congenital malformation of the larynx and epiglottis. This malformation is congenital, and consists not only in an abnor-

mally narrow glottis opening, but the epiglottis is so disposed that its sides are rolled up and almost in contact, and the aryepiglottic folds draw the tip of the epiglottis downward and backward, covering the superior opening of the glottis. Thomson has shown, as has also Luschka, that the infantile epiglottis is gutter-shaped, and it is only at the tenth year of life that this infantile type of epiglottis is lost and the adult type substituted.

In the cases of laryngeal stridor in which a postmortem has been obtained, the above abnormal anatomical condition was present. One of the first cases published was that of Lees, in which the child had suffered from laryngeal stridor and died of intercurrent diphtheria. In this case the aryepiglottic folds were apposed so that the epiglottis covered the larynx and a mere slit-like opening of the epiglottis was seen and through this breathing took place. A second case of laryngeal stridor in which autopsy was obtained was that of Refslund. This was the case of an infant two and one-half months of age, dying of intercurrent pneumonia. Here the larynx resembled closely that of the case of the present writer, whose case also died of intercurrent pneumonia, and in which the lateral borders of the epiglottis were apposed so as to form a mere slit which closed in the superior opening of the glottis and through which respiration took place.

The third autopsy obtained was a case of Variot in a child twenty-two months of age, dying of scarlet fever. In this case the stridor had existed from birth and there was an epiglottis deformed, though to a lesser degree than in the two preceding cases. Thus, in those cases of laryngeal stridor in which an autopsy was obtained there existed a distinct anatomical malformation of the epiglottis and superior opening of the glottis or larynx quite sufficient to account for the symptoms, and in one case the infant was two and one-half months old (Refslund), so that there could in this case at least be no question as to a postnatal factor in the production of the marked deformity.

Recently, Thomson and Turner, of Edinburgh, have taken up the subject of laryngeal stridor and elaborated a theory in which the "primary element in the causation of this condition is a disturbance of the co-ordination of the respiratory movements, probably due to some developmental backwardness of the cortical structures which control them." In their monograph, the authors have shown that the infantile epiglottis has a tendency to gutter-

shaped formation, and any disturbance of the rhythm of respiration would intensify the tendency of the lateral aryepiglottic folds to curl the sides of the epiglottis toward the median line, thus producing the malformation seen postmortem in cases of laryngeal stridor. Their theory is a combination of the nervous element with the anatomical deformity as a sequence of this nervous incoordinate breathing. These authors took the larynx of newborn infants and actually produced by means of suction on the trachea below the larynx a condition of the epiglottis closely simulating the anatomical condition found by Lees, Refslund and Variot. Thomson and Turner surmise that the anatomical deformity of the epiglottis found in the postmortems of cases of laryngeal stridor was caused by the prolonged action of the irregular respiration, as shown in their experiments. However this might appeal to us by its ingeniousness, the theory does not account for the cases like that of Refslund, in which the larynx was inspected two and one-half months after birth, scarcely time enough to have effected such marked anatomical changes as were seen in this epiglottis postmortem. Recently, the thymic origin of laryngeal stridor has been presented by Avelis, and more enthusiastically by Hochsinger. Hochsinger, by means of X-ray pictures, established to his satisfaction an enlarged thymus in some 23 cases and in these cases laryngeal stridor was present. Hochsinger even goes so far as to revive the term "thymic asthma." This theory is not supported by anything but clinical life study of cases. In addition to the X-ray, Hochsinger has made a study of the percussion of the thymus, and has, to his mind, verified the organ as enlarged, thus confirming the X-ray pictures.

We will not attempt at the present day to analyze the data of Hochsinger, which may or may not be confirmed by future studies. It is our object to record a case unique in itself and one of exceedingly rare occurrence. The case occurred in my hospital service and died there while presenting the symptoms of an exaggerated form of laryngeal stridor. The exaggerated symptoms occurred in connection with the dyspnea caused by the pneumonia from which the child suffered. The autopsy, which was complete, enables the author to add evidence to the anatomical theory in the causation of the symptom complex of laryngeal stridor.

W. H., male, one year, the youngest of eight children. No history of miscarriage in the mother; parental history negative.

Baby partly breast-fed. When six months of age had bronchitis; baby could sit up and say mamma and papa. Has no teeth and has signs of slight rachitis. Weighs eighteen pounds.

Since the age of three weeks, the parents have noticed that the breathing was noisy and that there was a crowing noise with the breathing. The child has been taken on this account to many clinics. At the age of five months the condition became more marked or worse and has persisted up to the time of the present illness.

Present Illness.—Ten weeks ago infant became ill with an attack of convulsions, following which there came fever and a discharge from the left ear which lasted a short time; since then the child has not been well; for the past two weeks child has been quite ill and the breathing rapid and with crowing character. There were several convulsions during the illness. There has been a cough, followed at times by vomiting.

Status.—On admission, the child appears to be well nourished; no teeth; signs of slight rachitis. There are heard on entering the room loud, noisy crowing or sawing inspiratory and expiratory sounds; otherwise child is not suffering from cyanosis or signs of laryngeal stenosis. There is little, if any, retraction of the suprasternal notch, though there is great retraction of the diaphragmatic groove. The noisy crowing respiration is the prominent feature of the case and can be heard across the room. There are sudamina on the body. Nothing in the fauces, but an attempt to look into the fauces is always followed by a stoppage of respiration and a loud inspiratory crow similar to that which is heard in laryngismus stridulus.

Thoracic Signs.—The loud breathing is heard over the chest, but there are distinct dullness, crepitant râles and increased vocal resonance at right base of the lung. There are also patches at the left apex and right apex behind of dullness, with crepitant râles. The remainder of the examination negative. A stethoscope placed over the larynx confirms the supposition that the crowing sound is laryngeal.

Diagnosis.—Congenital laryngeal stridor. Acute disseminated bronchopneumonia.

Subsequent History.—The crowing stridor continued, the frequency of the respirations increased from 58, which it had been, to 70 a minute, the pulse 130—temperature ranged from 103° to

105° F., and the child died in convulsions with increasing signs of dyspnea, cyanosis and heart failure, twenty-four hours after admission to the hospital.

Autopsy revealed the lungs to be the seat of extensive disseminated bronchopneumonia, the right lung being most affected in its lower lobe. The thymus was large and weighed 25 grams. The epiglottis and larynx showed that the child had a condition exactly similar to the cases of Lees and Refslund. The epiglottis was curved backward and lay over the superior opening of the larynx. The lateral borders of the epiglottis were in contact, leaving a slit which varied from half a millimeter in its greatest extent from the tip of the epiglottis to a millimeter and a half at the arytenoid cartilages, where the space between the aryepiglottic folds was a little wider than above. The aryepiglottic folds were almost in contact and were thin and membranous. The larynx was slit open and presented nothing pathological, except that before the larynx was split open an attempt to introduce the smallest size one-year intubation tube into the box of the larynx between the cords could not be carried out without great force, and this was not attempted for fear of injuring the tissues for future study. The opening of the larynx, therefore, was narrower than normal. There was no membrane, no inflammation, or swelling, no cicatrices, either above, on or below the cords.

There are points of interest about this case which must be emphasized. The diagnosis was not difficult. Both the laryngeal condition and the condition of the lung were correctly surmised by the author before death. The peculiar breathing, though exaggerated and accelerated, was classically like that of many cases of stridor which had come under his notice.

A careful percussion of the thymus during life failed to give any positive data and still the thymus was found large postmortem. The enlarged thymus would no doubt be looked upon by some as corroborative of the thymus theory in its influence in the production of the symptom complex of laryngeal stridor. It must not be forgotten, however, that the thymus in this case in no way was demonstrated to press on the trachea and that the anatomical conditions of the larynx and epiglottis fully accounted for the production of the stridor. The thymus in the author's experience is found to be enlarged in many diverse conditions. In his studies on the thymus, made in conjunction with the Jacobi monograph

Posterior view of the epiglottis and larynx in Dr. Koplik's case of laryngeal stridor. The epiglottis curves back over the larynx; the ary-epiglottidean folds are closely approximated, so that the whole superior opening of the larynx is covered in by the gutter-shaped epiglottis.

on the subject, the thymus was found as heavy as 30 grams in children who had met death under the most diverse conditions, including pneumonia. Enlargement of the thymus is, therefore, not an uncommon postmortem finding. During life, also, many lymphatic children no doubt have an enlarged area of dullness over the thymus, or an x-ray may show an extended shadow, and yet it would be difficult to say whether such a thymus is not rather the result of, than the cause of, the pathologic state of the patient; we know very little of the function of the thymus. Why multiply the diseases traceable to its enlargement without at least an inkling of its function?

Laryngeal stridor may occur in conjunction with a large thymus, but if there coexist such marked distortion anatomically of the epiglottis and larynx, as in the cases of Lees, Refslund, and that of the author, it would be going out of our way to draw the thymus into the picture. The theory of Thomson and Turner also needs more confirmation in view of the fact that in four consecutive autopsies, Lees, Refslund, Variot and that of my own case, the anatomical anomalies are fully sufficient to account for the symptoms. Until an autopsy reveals a perfectly normal larynx and epiglottis in a child in whom laryngeal stridor coexisted during life with a large thymus, we would be unwilling to entertain either the thymus theory or the theory of nervous incoordination of Thomson and Turner in its production.

692 Madison Avenue.

DISCUSSION.

DR. MORSE.—I would like to mention in this connection a case symptomatically like congenital stridor that I saw last winter. An examination showed that the larynx and vocal cords were normal and we thought we made out an enlarged thymus. It seemed apparent that death would result unless something was done. Dr. Murphy, therefore, removed a piece of the sternum and pulled up the thymus. When the thymus was pulled up the breathing immediately became better, but became difficult again as soon as it was allowed to fall back. The thymus was then stitched up into place. The symptoms recurred in a few days, however, and the child died of general weakness. I think that this is the first time that this operation has been deliberately undertaken for the relief of dyspnea supposedly due to hypertrophy of the thymus.

DR. GRIFFITH.—I think I recall reading the history of a case, perhaps not of the same nature as Dr. Morse's, in which the thymus gland was elevated in order to relieve the pressure of it upon the trachea.

I do not know whether Dr. Koplik means to include his case in the group known as thymic asthma; I suppose not. Writers seem disposed to divide cases of laryngeal stridor and spasm of the larynx into several groups. First, those due to lymphatism, of which the enlarged thymus is a symptom only rather than the cause. It is questionable in these cases whether the apparent laryngospasm is really such, or is an evidence of heart failure. Second, stenosis in which an enlarged thymus gland produces the symptoms by pressure. Cases of this kind are certainly quite unusual. Third, the cases of congenital laryngeal stridor, which are certainly distinct from the other two forms.

DR. MCCOLLOM.—I have been very much interested in this specimen because it shows that we cannot tell what difficulty we shall meet when we attempt intubation. I also want to say that in the majority of cases of laryngeal diphtheria we shall get negative cultures. That is an important point because we must not wait for positive cultures in those cases; the membrane is so far down that we cannot reach it with the culture wire. I emphasize this because I have had so much difficulty to make physicians understand that we must not wait for positive culture reports. Bacteriology is all right, but it must not entirely rule the clinician.

DR. LA FÉTRA.—In regard to the pressure of the enlarged thymus upon the trachea, most of the cases carefully studied show that the thymus does not make any pressure upon the trachea itself. There have, on the other hand, been a few cases examined where it was shown that the trachea was deformed or flattened by the pressure of the thymus gland. In these cases the dyspnea ceased when more room was made for the thymus.

DR. ABT.—I would like to ask Dr. Koplik whether this is an unusually heavy thymus. It seems to me I have seen glands as heavy as that which cause no symptoms whatever. If I remember properly, it is said in the Gerhardt Handbuch, in the Jacobi article, that a thymus of 30 grams may exist without causing any symptoms.

DR. KOPLIK.—That is correct, and you may remember that I myself weighed those glands for Dr. Jacobi. In regard to the point made by Dr. Griffith, I agree with him that the word thymic asthma should be held in abeyance until we can prove the pathologic nature of the condition. I am inclined to believe this thymus had little to do with the condition, for you find as large thymus glands in children who die of pneumonia and other diseases but who have never had laryngeal stridor.

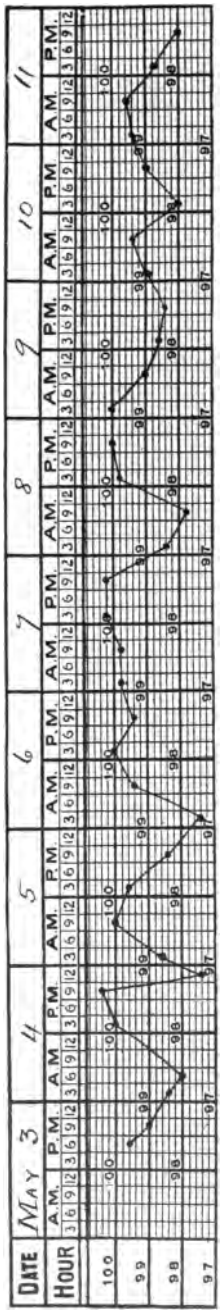
SCLEREMA NEONATORUM.

BY J. P. CROZER GRIFFITH, M.D.,
Philadelphia.

In February, 1897, an extremely interesting and characteristic case of sclerema neonatorum was brought to the wards of the Children's Hospital, where it came under my care. In looking up the literature of the subject later but 5 cases were found reported in detail in the United States, although a number of others were referred to incidentally by gentlemen taking part in discussions in some of our medical societies. Abstracts of the 5 published cases, and the full details of my own case were published in a paper in the Transactions of the Association of American Physicians, 1897, Vol. XII. Since that time there have been, as far as I know, but very few additional American cases reported. Dr. A. H. Davisson, of Philadelphia, gave the details of a fatal case before the Philadelphia Pediatric Society in 1902. A very typical instance was published by Browning in the *Journal of Cutaneous and Genito-Urinary Diseases* for December, 1900; another by Stillman in the *Journal of the American Medical Association* for April 25, 1903, and still another by Welt-Kakels in the *New York Medinische Monatsschrift* for 1904, Vol. XVI. Doubtless there have been others published which have not come to my notice, but certainly they are very few in number. The disease is therefore clearly rare, yet not to so great an extent as the data given would indicate. Cases undoubtedly occur, but are not reported. Dr. Richard Norris tells me, for instance, that there have been a number of cases under his care in the Preston Retreat in Philadelphia, and this is probably the experience of every large maternity.

The mortality of the disease is always extremely high. On this account the report of the following case will be of interest, as the child has now completely recovered. In some particulars the history is unfortunately lacking, yet there can be, I think, no question as to the diagnosis.

Elizabeth O'Neill, Irish, was admitted to the Children's Medical Ward of the Hospital of the University of Pennsylvania on May 3, 1905. The child was born March 6th. Her mother



TEMPERATURE CHART IN CASE OF SCLEREMA NEONATORUM.

died eleven days later either of tuberculosis or of sepsis; just which, it has been, as yet, impossible to ascertain. It is certain, however, that she had a cough for some time before labor, and had been in feeble health. She had had one miscarriage, and had borne two other children who were living and in fairly good health. The father was on a prolonged debauch at and after the time of his wife's confinement, and little can be learned from him. The aunt of the child took it under her care when it was ten days old, and the early history of the baby is obtained from her. It was born at full term, and was given condensed milk from the outset, this feeding being continued until the time it was brought to the hospital. It was a feeble and very thin infant, but with a strong cry. When it was eight days old the father said that the skin of the hips became as hard as the back of his hand. This continued, and when the child came under the aunt's care, the buttocks, too, shared in the hardness. Somewhat later the legs and back hardened in the same way. The baby had a tendency to hold itself with the back arched and the head retracted. There had been considerable vomiting. There is no history of convulsions. The skin of the buttocks is said to have been often bluish, but it cannot be learned that there ever existed any cutaneous discoloration which could be certainly attributed to an effusion of blood into the tissues.

On admission to the hospital the baby was found to be only

fairly well nourished, but showed no special signs of a marantic state. The fontanel was not depressed. There was no induration of the cheeks. The neck could be readily bent in any direction and exhibited no hardness. Over the external portion of the right shoulder, however, and extending down to the middle of the upper arm there was a large indurated area, hard and board-like, and apparently extending through the whole thickness of the skin. The skin as a mass could be moved but slightly over the muscular tissues, did not pit, and did not readily wrinkle. There was no discoloration. A similar condition existed in the left upper arm. The whole of the skin of the back of the thorax, as far downward as the middle of the dorsal region, was extremely indurated, illustrating the term sometimes used in describing this condition; *i.e.*, a "flitch of bacon," the whole skin moving almost in one piece. It could not be wrinkled except in coarse broad lines. The nates exhibited the same board-like hardness and this extended down the outer and posterior portion of the thighs, more than half way as far as the knees. There were also several small areas of induration in the calves. The feet, forearms, hands, and the anterior portion of the trunk showed no evidence of induration. There was no discoverable enlargement of the spleen or liver. The heart and lungs appeared normal, the child crying vigorously.

The baby was put at once upon a barley-water mixture, and an evacuant of the bowels. On the next day it was given a weak modified milk formula (fat, 2; sugar, 7; proteids, 0.75). General massage of the indurated areas was ordered daily, with the application of a hot water bag to the back. The food was borne well, and the formula was steadily increased in strength. There was an improvement, at first very slow, but afterwards rapid, in the condition of the tissues. By the 7th of May, four days after admission, improvement was so remarkable that I interrupted the massage treatment for a few days. On the 9th of May the child was exhibited before the Philadelphia Pediatric Society, and on that date the induration of the back was practically gone, and that of the buttocks and limbs was much less than formerly. The baby had lost weight for the first week or ten days, owing to the weakness of the food formula, but after that commenced to gain, and continued to do so steadily ever since. An examination of the blood on May 13th showed hemoglobin 70 per cent., red blood

corpuscles 3,220,000, white blood corpuscles 12,160. On the 19th of May, the history states that the induration had almost disappeared, except from the outer part of the hips and the areas in the calves, where it was still quite marked. Another note, upon the 8th of June, showed that all induration had practically gone, and that the baby was steadily improving in health, and was in excellent general condition.

Regarding the temperature early in the attack nothing is known. That while the child was under observation in the hospital is shown in the accompanying chart (page 28).

DISCUSSION.

DR. KOPLIK.—I have been interested in this paper because I have had some cases of sclerema neonatorum, both localized and general, and in looking up the subject I found that the percentage of recoveries is not so small, one author, Clementowsky, reporting at least 70 per cent. I have had a case of localized sclerema neonatorum in which one buttock and the whole thigh was affected. Massage and heat causes this localized form of sclerema to disappear, but I have never had a case of generalized sclerema get well with this treatment. I have had several cases of my own but have not published them because I have not been fortunate enough to get an autopsy and thereby add to the literature. Barker records a case exactly like Dr. Griffith's with a slightly raised temperature, which was fatal. In such cases there is always some complication to account for the rise in temperature.

DR. JENNINGS.—I might mention 2 cases coming under my observation, but which were, unfortunately, in an inaccessible part of the country so that I saw the children but once, a pair of twins, with a general sclerema simultaneously and both cases were fatal.

DR. JACOBI.—I would like to ask whether there was any pitting of the skin, as in edema?

DR. GRIFFITH.—No; there was no pitting of the skin. By the way, someone has used the term "flitch of bacon" to describe the appearance of the skin in this disease. It is a very appropriate one. Even had this child died an autopsy would probably have shown very little, inasmuch as this has been the experience with the autopsies which have been made.

DR. JACOBI.—The only way to learn anything about it, however, will be by autopsy.

CASE OF SEPSIS IN A NEWLY-BORN INFANT.

BY A. JACOBI, M.D., LL.D.,
New York.

G., male, 862 Park Avenue, was seen at 9 P.M., April 5, 1905, with Dr. Baran. Is the third child of the family. No miscarriage. First child was an eight months baby; died on the second day. Mother had been sick and under treatment for several months previously. Second child was delivered by Dr. Baran, and is in good health. No family disease, particularly no hemophilia.

History.—No written records were kept. The following history was elicited from the physician: Nothing was noticed until the fourth day. Then heavy uric acid infarctions were discharged. They lasted until the eighth day: recommenced on the ninth and lasted to the tenth day. Urine was pale on the eleventh. No examination was made. Quantity fair. Circumcision on the eighth, with no accident. Purpuric spots of small size were seen on the extremities on the ninth day.

Hematuria appeared on the twelfth and continued. On that day a consultant was called in. He found what has been described, and both kidneys swollen. Is reported to have diagnosticated tumors of both kidneys.

The cord fell off on the fifteenth day, April 4th. Was seen by me on the sixteenth, April 5th, 9 P.M. Air of the room good; window had been kept open; bedding clean; plumbing appears to be in order. Mother in fair health; sitting up; has no fissures in her nipples. No history of tuberculosis, or syphilis. Baby still weighs nearly six pounds; is said to have lost considerably. Mouth and nose normal; lips dry; somewhat fissured in the corners. Ears appear negative. No diarrhea. No malformation. Purpuric spots, small and large; some with slight elevation of the surface, over chest and epigastrium; some on face, shoulders, arms, fingers. Some painful livid elevations (suggesting the presence of pus in the deeper tissue). Icteric discoloration not noticeable in gaslight; is reported to be trifling. The liver large, as usual at that age. The spleen was not felt; percussion negative. The right kidney was not felt. The left kidney felt like the size

of a hen's egg, hard and smooth. Respirations about 60; pulse 200; temperature 104.5° F. Heart negative. Umbilical stump has some bloody oozing; is covered with some boracic acid, with which it has been dressed all along. The condition of the child appeared to warrant no close examination of the lungs, nor of the blood; no vein being in view or accessible under the circumstances. The baby died the next day.

Autopsy at 9 P.M., six hours after death. Surface as described in the living; some of the spots paler; some more livid. A moderate amount of serum, tinged with blood, in the pericardium. On it numerous petechiæ. Heart negative; thymus small, negative. Four of the lobes of the lungs have disseminated hemorrhages; some quite superficial, pleural and subpleural; some infarctions, mostly triangular of $\frac{1}{2}$-$\frac{2}{3}$ cm. in depth. Some atelectatic places in both sides posteriorly. Peritoneum holds a few ounces of blood-tinged serum, and shows a few petechiæ on the abdominal wall. Both costal pleuræ covered with petechiæ, and a few extensive extravasations. Liver as large as normal; negative. Umbilical vein and ductus Arantii, normal; not ulcerated. Spleen small; negative. Stomach exhibits circumscribed blood points in the mucous membrane. Many extend down to the submucous tissue. These changes are mostly found in the pyloric part.

Umbilical stump large; slightly eroded; covered with a scab of coagulum and boric acid. The pelvic connective tissue is black with blood. Both adrenals small; rather more so than normal.

Left kidney enlarged to almost twice its size; dislodged downwards from 4 to 5 cm.; capsule penetrated with blood; some clots between capsule and kidney; no open blood vessel found; capsule also thickened with fat. The upper part of the kidney forms a black, almost uniform looking mass, which so swells the tissue that fetal lobulation becomes indistinct. The right kidney is similarly changed, but to a far less degree. Section of the left kidney exhibits some small uric acid infarctions which are still held in the pyramids.

A few points are of unusual interest:

1. Uric acid was discharged in large quantities from the fourth to the eighth day; then again from the ninth to the tenth. Small hemorrhages, with or without secondary nephritis, are not very rare after uric acid infarction, but the suspicion that the foreign bodies might have caused the hematuria was soon dismissed.

2. It is certain that almost every floating kidney found in early age is congenital. As this baby had been lying down all the few days of his life, the increase in size should not be charged to the dislodgment of the left kidney.

3. The diagnosis of intra-abdominal tumors, until it be quite positive, should be suspended even in infants and children in whom intestinal contents are rarely misleading. Besides, what we feel inside is exaggerated by the mass at least of abdominal wall which has to be grasped on both sides of the questionable body. The left kidney *was* enlarged by hemorrhage, and was abnormally accessible, and the tumor of a kidney might be suggested by the findings. Still, very few tumors of a kidney ever bleed. Carcinoma does bleed sometimes; sarcoma very rarely; calculi in later life; tuberculosis not in the newly-born; cysts and hydronephrosis not at all.

4. The bacteric cause of this sepsis is not known; nor can we know the mode of its invasion. The amniotic liquor and the milk and lochia of the mother should not be accused as long as she was well and other causes cannot be found. The skin exhibited so many changes that its condition one or two weeks previously can only be guessed. The lips were sore at a late date. The umbilical stump was sore and bleeding. The cord had not fallen off before the fourteenth day; invasion is quite possible during that long time of the cutting of the cord (even the very tissue of the cord, unchanged, may admit microbes, or toxins); and boracic acid is probably not a sufficient antiseptic to be applied as a protection to a vulnerable surface like that of the navel.

DISCUSSION.

DR. HOLT.—I saw this case when the child was living. It was obscure then and I do not know that it is entirely clear to my mind now. Dr. Jacobi has given an excellent description of the specimens but has not pictured the wretched condition of the child. It weighed something like 7 or 8 pounds at birth. When I saw it the temperature was subnormal and all symptoms indicated a severe grade of inanition. The only striking thing found when I examined the baby was this tumor felt in the region of the left kidney. It was as large as a lemon at that time. The urine was bloody. The mother had practically no milk and the child was nursing on dry breasts.

I was puzzled as to the nature of the disease, but thought prob-

ably there were uric acid infarctions and a secondary acute hemorrhagic nephritis. The diagnosis of a neoplasm was also considered as possible. In view of the autopsy findings, sepsis seems to be the only explanation, and yet it is hard to see why one kidney should have been more affected than the other.

DR. HUBER.—In this connection, I should like to read the report of a case of sepsis in a very young infant, the point of entrance being the integument.

ACUTE EMPYEMA IN AN INFANT FIVE WEEKS OLD DUE TO STAPHYLOCOCCUS INFECTION.

Baby A., male, born April 5, 1905; normal delivery; weighed at birth six pounds; no tubercular or specific trouble. A few days after birth, small superficial pustules were seen upon various portions of the body. Successive crops appeared during the following four weeks. The circumcision wound and umbilical stump did not present any abnormality. When child was eleven days old, two small furuncles appeared upon the scalp, these healed kindly in about ten days. Nothing abnormal in mouth, nares or about the ears. During this time baby took both breast and bottle; its general nutrition was fair; stools greenish.

May 3d cough appeared, slight temperature was noticed; three days later temperature 102° F. Cough persisted and temperature continued, ranging between 100° and 104° F., until May 10th, when fever subsided. Respiration was accelerated, 50-60-70 per minute. On the eighth day (May 11th) some dullness was noticed on the left side posteriorly, which gradually became more pronounced, and at the end of the second week it was absolutely flat. During the second week the temperature was 99.5°, morning, and 100° to 101.5° F., afternoon. General condition better than during first week; breathing rapid and superficial, but not labored. Edema was observed about this time (urine negative); heart's action good throughout the course of the disease.

On the fourteenth day of his illness, the exploring syringe revealed pus. The bacteriological examination showed staphylococci only. The same afternoon the child was operated upon by Dr. Ladinski (simple incision and drainage with tube). The subsequent dressings were made by Dr. Rabinowitz. Recovery was uneventful; the wound had practically healed by June 12 1905.

In going over the points of the history, we find that in all probability the infection took place through the tender integument in some one or other of the numerous pustules present. The umbilical and circumcision wounds both had healed without any trouble; furthermore, there were no manifest lesions in the

mouth, nose or ears. The pus did not contain any pneumococci or streptococci, only staphylococci. The purulent collection in the left pleura must be regarded as the final onslaught of the staphylococcus infection, originating in the skin. Fortunately the process ran a subacute course, and the final result, in spite of the tender age, was very satisfactory.

DR. KOPLIK.—Of great interest, and bearing directly on Dr. Jacobi's case, I would relate a case which I saw in consultation. The child was nine days old and in perfect health up to the time of a ritual circumcision. The parents informed me that the operator used a knife that was not bright and was supposedly not clean. The parts were sucked to stop hemorrhage. Six hours later the child developed a hemorrhagic swelling about the wound and petechiæ all over the body. When I saw the child the next day it was dying, only twelve to sixteen hours after the operation. The body was black and blue as a result of hemorrhages and looked as if it had been beaten. The spleen was enlarged, and there was marked dyspnea. I mention this case to show how rapidly sepsis may develop through the introduction of streptococci into a fresh wound from the mouth. May not a similar factor have entered into the case recorded by Dr. Jacobi?

DR. EDSALL.—Dr. Jacobi may be interested to know that at the last meeting of the Philadelphia Pathological Society Dr. David Riesman reported several cases in newborn infants in which the chief lesion was hemorrhage into the kidneys, resembling exactly the kidneys that Dr. Jacobi has shown. In one or two of the cases he found a general staphylococcus infection. The lesions were limited solely or almost entirely to the kidneys, and Dr. Riesman described the condition as probably a pathological entity.

DR. SAUNDERS.—I think it is pretty well established that any infection in the newborn is liable to take on a hemorrhagic character. Whatever the organism may be, the infection is likely to be hemorrhagic and fatal.

This case reminds me of one that was seen some years ago and remains a puzzle to this day. It was a newborn infant and the condition was marked by high temperature. At autopsy one kidney was found very much enlarged and looked like a spleen. I had cultures made from the kidney, but nothing was found. I have also in practice met with 2 or 3 cases in which I was tempted to make the diagnosis of rapidly growing neoplasm of the kidney. One of them proved to be a protracted case of malaria; I had given a bad prognosis, but the child is now large and healthy.

I should like to know from the members of this Society if in an acute fever they have been able to palpate the kidneys, in-

creasing in size like the spleen in typhoid fever, and yet the child get well.

DR. JACOBI.—My object in reporting the case was to point out the danger of diagnosticating a tumor under such circumstances. Dr. Holt had been under the impression that there was a tumor in this case, and as he was mistaken, I thought it worth while to report the case in connection with that possibility.

Secondly, it is a case of sepsis undoubtedly not of the usual kind. Most of the cases have been decidedly umbilical. Sepsis may be the result of absorption from the stump; in the large majority of cases we find some traces in the umbilical vein. This vessel was absolutely normal in this case. The first appearance of anything abnormal was in the lungs and these were filled with infarctions. Then there were hemorrhages in the skin and under the skin, in both kidneys and on the pleura, as we may see in any extensive sepsis. The question was, Where the sepsis arose and when? When I saw the case the skin and mucous membranes of the nose and mouth had undergone changes from day to day. Sepsis may have originated from them, or through the tissue of the umbilical cord long before it fell off.

Sepsis is more common, I believe, than many of us have thought. Babies die after a few weeks of what is called pneumonia. If babies were examined more carefully and temperature records kept, we might find sepsis more frequently. The infection may be a sudden one. According to a number of observers it takes from six to eight hours from the time of establishment to the showing of the first symptoms. But in Dr. Koplik's case it is possible, of course, that the baby was septic before the circumcision, and, in the case I saw, there may have been something before that I did not know of.

A CASE OF STENOSIS OF THE PYLORUS IN AN INFANT.

BY J. CROZER GRIFFITH, M.D.,
Philadelphia.

It has been interesting to note the manner in which stenosis of the pylorus in infancy has come into prominence in the medical world. Although Beardsley in 1788 (reprinted by Osler in ARCHIVES OF PEDIATRICS, May, 1903, p. 355), reported a case which was certainly of this nature, little further was contributed upon the subject until Hirschsprung's article in 1888 (*Jahrb. f. Kinderh.*, 1888, Vol. XXVIII., p. 61) directed renewed attention to it. Although reports of cases then began to appear with increasing frequency, it is safe to say that until within a very few years a large number perhaps the majority of physicians were disposed to accept the existence of the condition with great reserve, and certainly to deny the possibility of making a diagnosis. The continued and rapid multiplication of reported cases has now removed all doubt as to the existence of the disease. Regarding the possibility of the making of a diagnosis, not confirmed by autopsy, there is greater question. Nevertheless there are a large number of cases which have recovered yet which have exhibited in the most characteristic way the symptoms present in the fatal cases which have been confirmed by autopsy. My purpose now is to present another instance of this nature.

E. S., male, was born December 25, 1903. He was a well nourished child, weighing ten pounds. In the first two days of life he was constipated, but from the third day, when the milk secretion began, slight diarrhea was present, a watery stool occurring within an hour after nursing. He had been gaining normally in weight. On January 10th, when sixteen days old, he appeared to have indigestion and vomited. On January 12th he seemed stupid, had no appetite, was constipated, and vomited occasionally. There was no fever. I saw him for the first time on January 14th. He was still very drowsy. His constipation had been relieved only by the administration of castor oil by injection, which brought away dark colored mucus. I advised that he be taken temporarily from the breast, which appeared to disagree with him, and given barley water. On the afternoon of January

14th, being now three weeks old, frequent vomiting began, all food and medicine being rejected. This condition continued all night. During this night peristaltic movements in the region of the stomach were clearly seen for the first time, a wave passing from left to right, and the stomach appearing to be dilated.

Vomiting continued from this time, the child emptying his stomach four or five times, and sometimes seven or eight times in the twenty-four hours. I saw him again on January 18th, and made the following note:—Vomiting has continued from four to eight times in twenty-four hours. No gas is passed by the bowel as far as has been noticed. Once or twice a day a dark greenish or brownish viscid mucous stool is passed containing considerable bile, following or independent of intestinal douching, but no fecal matter whatever has been discovered. The abdomen is soft; not distended. The stomach is evidently dilated and extends below the umbilicus; active peristaltic movements of it can be seen, the wave moving slowly from left to right. Sometimes only one wave is visible, at other times two or even three can be seen at once, giving the appearance of tennis balls rolling slowly after each other under the skin. This movement is particularly active when the child is approaching an attack of vomiting. Lavage of the stomach is followed by cessation of vomiting, but after a time, as the organ seems to become full, it empties itself forcibly, either in one forcible gush or in repeated small vomitings. It would appear that little if any food passes the pylorus. There is little, if any, fever. The pulse is of fair strength.

For ten days from January 14th the condition continued with but little change, the vomiting occurring less frequently, but no fecal matter whatever appearing in the stools. Various forms of feeding were tried during this period, a mixture of egg-white and whey given in very small quantities frequently repeated appearing to be best tolerated, but all other milk food produced considerable pain. There had been the loss of a pound in weight during the week preceding January 16th, but after this the weight had varied practically not at all.

After January 24th, small fecal masses began to appear at times in the stools, which were passed from four to six times in twenty-four hours, and containing considerable mucus. The amount of normal fecal matter gradually increased, vomiting was much less frequent, and ceased entirely for a considerable time after February 5th; evidently some degree of stenosis still existed, the peristaltic movements being often visible, and the

stomach being decidedly dilated. Lavage was still practised daily, or every other day, according to indications; the diet continued to be of the lightest nature, weight remained unaltered, and the general condition on the whole good. The administration of Keller's malt soup was commenced after about March 5th, and agreed for a time, and a gain in weight commenced at that time.

The condition slowly improved, the child requiring, however, very careful management with changes in diet from time to time. Vomiting returned at intervals; the stomach was still dilated; the peristalsis was often active. Up to about May 1st it seemed evident that some degree of stenosis persisted, but that the trouble now was largely one of impaired digestion. Wet nursing was commenced shortly after this date, and the child continued to improve. He is now a healthy, well developed infant.

PHOTOGRAPH OF DR. GRIFFITH'S CASE OF PYLORIC STENOSIS, SHOWING PERISTALTIC WAVES.

A photograph taken at about the age of three months (March 25th) shows well the position and appearance of the peristaltic action, the outlines of the three waves having been sketched on the skin in the position often seen. (See above illustration.)

This case was at first a puzzling one on account of the distinct evidences of indigestion which were present at the onset, and which continued after the more characteristic symptoms of stenosis had disappeared. It is possible that the symptoms developing on

January 10th were those of indigestion merely, and that the actual stenosis did not occur until the afternoon of January 14th. It seems equally possible, however, that January 10th marked the onset of the stenosis. The cause of the obstruction in this case was evidently chiefly spasm, the action of which was probably aided, however, by swelling of the mucous membrane, dependent upon the irritated state of the whole gastrointestinal tract. Possibly, too, some slight degree of hypertrophic change was present. The persistence of symptoms appeared to support this latter view.

I shall make no special reference to the literature of the subject, as this has been well done by many writers, particularly in the papers by Dorning and by Shaw and Elten, read before this society last year (Transactions American Pediatric Society, 1904), and in the excellent article by Wachenheim (*Amer. Jour. Med. Sci.*, April, 1905, p. 636) recently published.

It is customary to divide cases of stenosis into two classes. First, those in which the obstruction depends upon an actual congenital anatomical lesion; viz., a hypertrophy of the tissues of the pyloric ring with consequent narrowing of the lumen. Second, those in which the stenosis is spastic in nature. It seems perfectly possible that there may be a combination of the two conditions; that with a slight degree of hypertrophic stenosis there may be a temporary spasm, such cases being capable of recovery. It is also possible that temporary swelling of the intestinal mucous membrane may aid in producing closure in either of the classes. We can do little more than theorize on this subject. My own case as I have stated supports the theory that a stenosis chiefly spasmodic may be aided by either of the two other conditions.

Two interesting practical points are to be deduced from what is already known of the subject. First, that even undoubted hypertrophic stenosis is capable of being cured by operative interference, 27 per cent. of recoveries according to Wachenheim. Second, that as, however, the mortality is high, we should entertain the possibility of the disease being spastic merely and capable of recovery without surgical aid. To determine how long to wait without delaying so long that operation must be only hopeless in the infant's weakened condition can be determined only by the individual physician in the individual case. The case which I have reported exhibited a stenosis apparently complete for ten days and more or less obstruction for weeks longer. Operation was postponed because the child seemed to tolerate the affection fairly well, and to be maintaining his weight satisfactorily.

TWO OPERATIVE CASES OF PYLORIC STENOSIS IN INFANTS.

BY THOMAS MORGAN ROTCH, M.D., AND MAYNARD LADD, M.D.,
Boston.

The following 2 cases of pyloric stenosis are reported as being of interest, from the fact that in 1 complete recovery followed after operation, and in the other an interesting postmortem examination was made.

CONGENITAL PYLORIC STENOSIS IN AN INFANT THREE WEEKS OLD. OPERATION. RECOVERY.

This case of pyloric stenosis is worthy to be reported for several reasons.

(1) It is the youngest case in medical literature to be operated upon with complete recovery.

(2) There is only 1 other case reported at this early age. This was one of Thompson's 6 cases which was operated on by Stiles and died in thirty-six hours.

(3) It exemplifies in an absolutely typical manner the clinical features of complete pyloric stenosis.

(4) It contains very strong evidence that complete pyloric stenosis may exist as a congenital condition.

This case was seen by Dr. Ladd in consultation with Dr. Henderson, of Reading, Mass., and with Dr. John C. Munro, of Boston, to whose courtesy we are indebted for the privilege of placing it on record.

D. P., a boy, was three weeks old. He was the second child. The first baby was nursed with absolutely satisfactory results by the mother. The mother had an abundance of milk and was of a temperament well adapted to nursing. The baby weighed at birth ten pounds when dressed, and appeared perfectly healthy. The labor was normal and the mother made a quick convalescence. The baby was put to the breast at the usual time and at two hour intervals. About the time the milk came freely, vomiting began

and had continued, not a single feeding being retained. The vomiting occurred usually soon after the nursing, sometimes as much as an hour afterward and was generally expulsive in character. There was no bile in the vomitus. The breast or bottle was always taken greedily, the baby acting as if he were starved. The movements were very small in amount, green in color, and consisted mostly of mucus and a few small specks of matter which may have been fecal, but the amount was so small as to indicate that practically a complete obstruction prevented the passage of the food into the intestines. There was no difficulty in deglutition.

Dr. Henderson had tried various dilutions of the breast milk, condensed milk and water, a 4 per cent. solution of milk sugar, and a 2.5 per cent. solution of lime water. He also had experimented with amounts ranging from one drachm of these various mixtures to 2.5 ounces. The result was always the same—the feeding was not retained. On a few occasions the baby took as much as 2.5 ounces at a feeding which showed that the capacity of its stomach was quite as large as normal, if not larger.

The physical examination showed, so far as could be determined, a child normal in all respects except for a considerable degree of emaciation. It probably did not weigh more than six and a half to seven pounds. The abdomen could not be satisfactorily palpated as the baby cried vigorously when disturbed. Under ether, moreover, nothing abnormal could be felt in the region of the pylorus, but the outline of the distended stomach could be seen through the relaxed abdominal wall. The pulse was 200. The temperature and respirations were normal.

The diagnosis of pyloric stenosis was made from the clinical picture and immediate operation advised. It was quite obvious that the food was not principally responsible for the symptom of vomiting. The practical absence of fecal movements showed that whatever nourishment was obtained could be only a small amount which might come from direct absorption from the stomach itself. This was not enough to prevent rapid emaciation and loss of strength. The indication for exploratory laparotomy was therefore quite clear.

For the next forty-eight hours while the parents were making up their minds whether to risk an operation, it was suggested that a modified milk calling for a 2.00 fat, 5.00 sugar, and 0.75 proteids should be prepared, peptonized for one hour and given by

rectum every four hours, two ounces at a time. At the same time teaspoonful doses of breast milk were to be given by mouth every hour. The enemata were retained and appeared to give the baby more strength. The teaspoonful doses of breast milk were retained until about one and one-half ounces had been taken and then the whole amount was expelled as usual by vomiting. After that teaspoonful doses of brandy and water were given every hour and were presumably directly absorbed, but larger quantities were vomited. The case was operated on by Dr. John C. Munro at the Carney Hospital. Operation under ether. Median incision. On opening the abdomen the stomach was found considerably distended with gas, and was dilated to about the size seen in a normal infant of three months, with a capacity of about four ounces. At the pylorus was an oval solid tumor approximately three quarters by three-eighths of an inch in diameter. It was situated high up under the liver, which explained the fact that it could not be felt before operation even under full narcosis. A posterior gastro-enterostomy was made with clamps and suture, a jejunal loop being used. The jejunum was no larger than a pencil, and by the time the various sutures were placed, three-fourths of the gut were taken up in the anastomosis. An opening easily admitting the finger tip was made. The patient was in fairly good condition at the end of an hour.

The subsequent history and after treatment of the case were as follows:—During the day of operation the patient was given brandy mx, salt solution ℥ss per rectum every three hours for two doses; then peptonized milk ℥i, and brandy mv per rectum every four hours. Sterile water was given in drop doses by mouth during the night.

On the following day, June 29th, there was a small movement of the bowels with gas. Rectal feeding was continued and peptonized milk was begun by the mouth, beginning with drop doses and increasing gradually. In the afternoon peptonized milk by mouth ʒiv every two hours was started. The bowels moved three times during the night. From this time on recovery was uneventful, the fecal movements on July 2d being normal in color and consistency. There was very little vomiting and the child gained in weight rapidly. Discharged well on July 17th. At the end of one year the baby was in health and physical development fully up to the standard of the average healthy infant.

PYLORIC STENOSIS IN AN INFANT FOUR WEEKS OLD. OPERATION. DEATH.

This case was admitted to the Infants' Hospital, Dr. Rotch's service, on January 2, 1905, with the following history:—Vomits a good deal after every feeding. Has never kept down a feeding. Bowels move only with enemata. Movement shown is simply a green, loose movement, with mucus and no fecal matter. Passes water once in five hours.

Physical Examination.—The infant was emaciated. The heart was normal. The lungs were negative. The abdomen was somewhat distended, but was soft and readily palpable. No enlargement of liver or spleen, and no tumors were detected. There was hyperresonance from just below the ensiform cartilage down to the umbilicus. Forty-five cc. of modified milk were given at eleven o'clock. At the time of the examination an hour later, on having the child sit up, the hyperresonance disappeared and was replaced by dulness extending to the level of the umbilicus, evidently from the fluid swallowed at eleven. Hyperresonance returned when the baby was placed flat on its back again. There had been no vomiting since the feeding at eleven. The rest of the physical examination was negative.

January 4th. Vomits occasionally clear fluid.

January 5th. Vomits more than on the first day. Peptonized milk was given by the rectum. Only boiled water was given by mouth.

January 6th. Operation by Dr. Stone.

Median incision from umbilicus upwards to just above the edge of the ribs. Wound carried through fascia to peritoneum. Peritoneum incised. Stomach found fully distended. Pylorus firm, making a mass about one inch long by three-eighths of an inch in diameter. Below the pylorus the large and small intestines were completely collapsed. The omentum was pushed to the left, the transverse colon pushed back and the first loop of the jejunum drawn forward into the wound. The lowest anterior portion of the stomach was withdrawn from the abdomen and seized with clamps; the jejunum being too small to clamp, was seized with hemostatic forceps at a portion farthest from the mesentery and approximated to the stomach. A running stitch including the peritoneum and muscles was carried along the posterior approximated surfaces. An eliptical piece of the stomach was incised and a longitudinal slit was made cutting the duode-

num in front of this stitch; a continuous silk stitch was carried around uniting these openings, and passing through all the layers. The posterior stitch was then continued around through the serous and muscular coats in front of the deep layer of stitches and tied at the starting point. The clamp and walling off gauze were then removed and the area washed off with salt solution. The stomach and the jejunum were returned to the abdomen. The omentum was tucked around the area. The incision was then closed with interrupted silk worm stitches through the entire abdominal wall.

During the operation which occupied about twenty-six minutes from the first incision till the last stitch in the abdominal wall was tied, the child's condition became very bad. Salt solution was given under the skin, and brandy minims 30. At the end of the operation the shock was extreme, the pulse was scarcely perceptible at the wrist, and the respiration were very irregular, jerky and at long intervals. Heaters were applied and the foot of the bed elevated. The extremities were rubbed with warm alcohol. Brandy minims 15 was given subcutaneously after the infant was put to bed.

January 7th, at about nine o'clock, the baby began to vomit a bright watery fluid and continued to do so during the day. One teaspoonful of whey was given every half hour up to ten o'clock and then every hour during the night. Nutrient enemata continued.

January 8th. Half a drachm of water was given every hour. The enemata were not well retained.

January 9th. Enemata omitted. Three teaspoonfuls of whey were given every hour and a teaspoonful of water every hour. The infant began to vomit again at 4 P.M., and vomited constantly during the night. Whey and water were omitted and the enemata resumed.

January 10th. Vomited very little. Became very weak and died about 11 P.M.

January 11th. Examination after death showed a healing incision one and three-quarter inches long above the umbilicus. No signs of inflammation. Adhesions of peritoneum with slight fibrino-purulent exudate on coils of small intestines and on edge of liver about the seat of the operation. Omentum adherent anteriorly. Pylorus very firm, admits probe two mm. in diameter easily. Coils of small intestines normally distended. The examination showed the cause of death to be a localized peritonitis

behind the anastomosis. There had been apparently no leakage and curds had been passed in the movements.

The emaciation and weakness rendered the child a most unfit subject for operation, but in spite of this vomiting was immediately relieved by the operation, the consistency of the stools changed, curds appeared in the movements and the small and large intestines became normally distended. The outlook for recovery would have been excellent had it not been for the peritonitis which was obviously the cause of the recurrence of the vomiting and death.

The operation and subsequent course confirmed absolutely the diagnosis and would encourage one to undertake earlier operation on a similar case before weakness and emaciation became so pronounced.

The medical and surgical aspects of pyloric stenosis in infants have recently been so fully discussed in elaborate papers, especially that of Dr. C. L. Scudder that it would but be repetition to bring them up again in reporting these cases.

It may however be said that while in the cases of complete pyloric stenosis there is seldom much difference in opinion as to the diagnosis and treatment, yet in the cases of incomplete stenosis, both congenital and acquired, judgments often differ. Certain general considerations should therefore always be carefully kept in mind as we ourselves have already met with a number of instances in which the surgeon was inclined to operate where a diagnosis of even partial stenosis was not justifiable.

We should recognize that a case of faulty feeding without any organic lesion may exactly simulate partial pyloric stenosis and also note that stenosis as a cause of vomiting is very infrequent in comparison with the innumerable cases of severe vomiting in no way dependent on organic disease, but on disturbance in digestion and metabolism.

We should also remember that while we cannot assume that pyloric thickening is absent because a mass cannot be felt, yet that in this class of cases the diagnosis should be arrived at only after a process of exclusion by careful experimentation with the food, following the general principles of modern scientific feeding. Even then in the majority of cases often only presumptive evidence can be obtained, but it may be enough to justify exploratory laparotomy.

DISCUSSION OF THE PAPERS OF DR. GRIFFITH AND OF DRS. ROTCH AND LADD.

DR. MORSE.—I have had the opportunity of seeing 4 cases of this disease. One of them has been reported by Dr. Townsend. Another one came into the hospital this spring when it was four weeks old. It began to vomit when two weeks old while taking breast milk, and though it was weaned and the food repeatedly changed, the vomiting increased. On the day it entered the hospital it passed a breast pin about one inch long and one-eighth of an inch wide, which had been missing for two weeks, so that at that time it must have had a pylorus that would permit the passage of such an object. There was nothing characteristic about the vomiting, which occurred ten or twelve times a day. The bowels were constipated, and the movements looked like meconium. A satisfactory examination of the abdomen was impossible until a week later because of rigidity of the abdominal walls; the stomach was then palpable below the navel, and there was visible peristalsis. This observation was repeated later, and an operation was advised. The advice was rejected for several days, and the baby was not operated upon by Dr. Murphy until it was six weeks of age. An enlarged and thickened pylorus about the size of a large cherry, was found at the operation. The striking thing to me, however, was the appearance of the intestines rather than that of the stomach. The intestine was not larger than a lead pencil anywhere. A posterior gastroenterostomy was the only operation possible. This was done, but the baby died in ten hours as the result of its extremely bad condition at the time of operation. The specimen has not yet been properly examined by section, but is now in the course of preparation.

In another case which I saw, a male child, also breast-fed, gained weight in the first few weeks, but began to vomit in the fifth week. Change in the feeding made no difference. In this case the vomiting occurred only once or twice a day, and large amounts were vomited at a time. The bowels were constipated and the discharges of a yellow color. I saw it first when eight weeks old. The abdominal examination was negative. As the symtoms increased steadily, it was operated upon by Dr. Bottomley in a similar way to the other case a few days later. It died ten days later.

Another case was seen at the Children's Hospital last summer. When it came in the tumor connected with the pylorus was easily palpable and about the same size as in the first case. Change in the feeding relieved all the symptoms of stenosis, although the tumor remained. The baby unfortunately developed intestinal indigestion and died of that without any recurrence of the symptoms of obstruction at the pylorus. I think this case might have recovered under medical treatment had the child's digestion been better.

DR. ABT.—I desire to refer to a case of pyloric stenosis which

came under my observation. I believe it is a variety somewhat different from that which has been commonly described in the recent literature. During the winter we admitted a little patient about three months old. He was being fed at the breast, but was brought to the hospital because he was vomiting continuously and losing in weight. We admitted the mother with the child because we wanted to continue the breast feeding if possible, and thought that by improving the nutrition and environment of the mother we would improve the quality of the milk. In spite of the breast feeding the child continued to vomit almost continuously. We weaned the baby and discharged the mother from the hospital. Examination of the child showed that it was greatly emaciated, there was no abdominal tumor to be felt, the stomach was moderately dilated, which was proved by inflating it with air as well as by the usual percussion methods. The baby was fed on artificial food; low percentage fat and proteid mixtures were tried, the stomach was washed out, the child was fed by the tube, but the condition remained unchanged. Very frequently the peristaltic wave with a recoil was observed in the region of the stomach; the wasting was continuous, and after four weeks of treatment we concluded to call surgical consultation. The surgeon agreed to perform gastroenterostomy, which was done with great skill, requiring eight minutes. The child, however, did not recover from the effects of the operation and died in twenty-four hours. The autopsy showed that the lumen of the pylorus was constricted its entire length. On making a slight incision through the wall of the duodenum and attempting to pass a probe upward through the pylorus into the stomach, it was noticed that the probe passed with great difficulty and could be made to enter the stomach only by using a great amount of force. A section of the pylorus was removed and submitted for examination. Prof. Hektoen reported to us that the specimen showed that there was an overgrowth of connective tissue, and that the bowel was constricted in consequence. It should be said, too, that there was no enlargement in the region of the pylorus such as is commonly observed in the cases of hypertrophic stenosis. The case then was undoubtedly one of congenital atresia of the pylorus.

I believe that in considering these cases we should divide them into three classes: (*a*) Spasmodic constriction of the pylorus; (*b*) hypertrophic stenosis, and (*c*) congenital atresia.

DR. ROTCH.—I have nothing to add to what I have already said, except that it would seem that the symptom of gastric peristalsis has been somewhat exaggerated, as it is often not diagnostic in infants. In a certain class of infants, especially in the gastric dilatation which is found in rhachitis, this peristalsis may be noticed without pyloric stenosis being present, and again where pyloric stenosis is present peristalsis may be absent.

DR. HOLT.—It seems to me we are greatly in need of deciding

upon what symptoms the diagnosis in this disease shall rest. Undoubtedly there are many reported cases that are not really the disease. The Germans lay great stress upon the peristaltic wave and it certainly is a valuable symptom, but personally I have not found it in all cases. There are some cases in which one who is familiar with the disease can have no doubt about the diagnosis after two or three days' observation. There are others in which, after weeks of observation, we must feel uncertain.

DR. GRIFFITH.—I do not think I can add anything which will make the matter of the diagnosis of the disease more clear. It seems to be necessarily very uncertain. I recall one case seen last spring in the Children's Hospital, where one of my colleagues was for weeks in doubt. The child began to improve promptly after a change of food which, by a mere accident, happened to suit it, for many changes in diet had previously been made. I recall also another case in a quite young infant, who certainly exhibited many of the cardinal symptoms of pyloric stenosis, including obstinate vomiting and marked peristaltic movements in the gastric area. This child recovered promptly after a wet nurse was employed.

THE USE OF CITRATE OF SODA IN INFANT FEEDING.

BY HENRY LARNED KEITH SHAW, M.D.,
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Twelve years ago E. A. Wright[1] wrote a short article on "The Possible Advantages of Employing Decalcified Milk in the Feeding of Infants and Invalids." In it he advocated the addition of sodium citrate to milk in order to prevent the formation of thick curds and suggested the possibilities of its use in infant feeding. No practical application seems to have been made of this suggestion until last year, when Poynton[2] published an article on "The Value of the Addition of Citrate of Soda to Cow's Milk in Infant Feeding," and a few months later Variot[3] wrote a brief account of the "Action thérapeutique du citrate de soude dans les troubles digestifs des jeunes enfants." Ausset[4] followed up the experiments of Variot, Poynton, and Wright, and obtained excellent results from this method of treatment.

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To convince myself of the claims of Wright and Poynton, I undertook a series of laboratory experiments, followed by a clinical trial, in order to test the value of this method.

We are not yet in a position to know with any degree of accuracy what happens chemically when the enzyme rennet is added to the milk. The calcium salts are necessary for the coagulation by rennet and this coagulation is retarded and even prevented in an alkaline medium. The acid curd differs chemically and physically from that produced by the action of rennet. Chapin and Southworth have rendered a distinct service by collecting the results of recent work on casein precipitation and pointing out the bearing on the problem of infant feeding. They show that the lime is combined definitely with the casein into what is termed calcium casein. The action of rennet converts the calcium casein into calcium paracasein. Calcium paracasein is a soft, tender clot, but when acted upon by the acids of the stomach—whether lactic or hydrochloric—it is converted into free paracasein and then into a new chemical compound known as hydrochlorid of paracasein. These curds are firmer and tougher and require strong digestive power for digestion. If the soft calcium para-

casein clot is not precipitated, the tougher, free paracasein clot will not be formed.

It is a well-known chemical fact that oxalates and fluorides will combine with calcium forming insoluble salts which are poisonous, and Wright suggested the use of citrate of soda to precipitate the calcium. Citrate of soda is little used in medicine and is classed among the unofficial drugs of the United States Dispensatory. It is a neutral salt, white, crystallizes in rhombic pyramids and has a saline taste without bitterness. It is non-poisonous and in large doses is a mild purgative. It is very soluble. It combines with calcium forming an insoluble salt—citrate of calcium.

To ensure uniformity in my laboratory experiments, 5 cc. of milk were used in each of the tests. To this were added the various solutions and just before the test tubes were placed in a water bath with a temperature of 40°C., two drops of liquid rennet were added and the time required for coagulation noted. All these tests were repeated, in some cases several times, and there was very little variation from the figures given below:—

	TIME REQUIRED FOR COAGULATION.	CONDITION OF CURDS.
5 c.c. milk.	30 sec.	solid curd.
5 " " and 1 c.c. ⅛% sol. cit. sod.	60 sec.	soft curd.
5 " " " 1 " plain water.	60 sec.	solid curd.
5 " " " 1 " ¼% sol. cit. sod.	90 sec.	fine curds.
5 " " " 1 " ½% " "	2½ min.	very fine curds.
5 " " " 1 " 1% " "	5 min.	very fine flakes.
5 " " " 1 " 2% " "	no react.	
5 " " " 1 " 3% " "	no react.	
5 " " " 1 " 4% " "	no react.	

A duplicate series of tests was made in which 5 drops of $^{n}/_{10}$ HCl were added and the only effect was that the coagulation took place a few seconds sooner. No reaction occurred when the citrate was added in a greater strength than a 2 per cent. solution.

The addition of plain water delayed the curdling, as has been pointed out by Chapin; but even when diluted five times the resulting curd was firm and solid. The addition of lime water, 1 per cent. solution of bicarbonate of soda, and 1 per cent. solution of phosphate of soda retarded the coagulation, but did not prevent it. When stronger solutions were added, no coagulation occurred until after the addition of $^{n}/_{10}$ HCl. This shows that rennet coagulation will not take place in an alkaline medium.

Citrate of potash added had about the same effect as the citrate of soda and probably could be used clinically for the same purpose. Citrate of magnesia hastened the coagulation owing to the fact that citrate of magnesia is an acid salt.

When a few drops of a 2 per cent. calcium chlorid solution were added to the uncoagulated sodium citrate tubes, the coagulation would take place almost immediately.

Sodium citrate had no effect on the acid coagulation on the addition of acetic acid. Citrate of soda in solution is neutral to litmus paper and only very slightly reduces the acidity of milk when it is added in a strength of 10 per cent. Lime water and bicarbonate of soda render the milk distinctly alkaline.

The same tests were made with boiled milk and the sodium citrate solutions below 1 per cent. were coagulated at longer intervals, but 1 per cent. solutions and over remained unchanged.

The curds in all the tests where plain milk was used alone or with the addition of water, were solid and jelly-like. The addition of weak solutions of the alkalies, while not markedly delaying the action of rennet, yet acted upon the curd so that it was precipitated into small, fine curds instead of a solid mass. Citrate of soda in small quantities had a marked effect in breaking up the curd into fine particles.

The explanation of the action of citrate of soda as advanced by Wright and Poynton is that the excess of lime salts in cow's milk is precipitated. No details are given of the chemical reaction that takes place. Variot says that a chlorid of calcium is precipitated, but as this is a soluble salt the explanation is hardly satisfactory.

Theoretically, the insoluble citrate of calcium should be precipitated. In order to ascertain if this were so I made some experiments with Dr. E. J. Wheeler, chemist to the New York State Department of Agriculture. We treated rather large amounts of milk with citrate of soda solutions and then filtered through ashless filter paper with the aid of a suction pump. The filter papers were dried, ignited and weighed. In no instance did we get an appreciable amount of ash and were forced to the conclusion that no precipitation in a chemical sense occurs, but that probably some unknown and undetected action or combination takes place between the sodium citrate and the calcium casein.

The clinical trial was made at St. Margaret's House. Since last November twenty-two babies have been on citrate of soda

mixtures. The length of time the babies were kept on this food was from three weeks to seven months and the average duration of feeding was about four months. Fourteen babies are still taking citrate of soda. These are all institutional babies who are more or less crowded in the wards and who did not get out of doors the entire winter. They were not picked cases and were nearly all undersized and underweight. The majority were suffering from gastrointestinal indigestion as shown by loss in weight, habitual vomiting and offensive, undigested stools. A number were typical cases of malnutrition. The citrate was given in some of these cases as a last resort when other methods had failed.

The following *résumé* gives a brief statement of the 22 cases:—

LOSSES.

CASE I.—Elsie R., six weeks old, was on citrate mixtures four weeks. Weight before taking, 8 pounds, 12 ounces; weight after, 7 pounds. Was a well-nourished breast baby on admission. Lost 8 ounces in two weeks on a whey and cream mixture. Was running a temperature while on the citrate. Died. Autopsy showed acute ulcerative rhinitis, bronchopneumonia. The gastrointestinal tract was normal.

CASE II.—Paul H., three months, was on citrate three weeks. Weight before, 11 pounds, 6 ounces; weight after, 10 pounds, 10 ounces. The child died in a convulsion two weeks after the food had been changed to whey and cream. Autopsy was negative.

CASE III.—Henry F., 11 months, was on citrate two months. Weight before, 18 pounds, 10 ounces; at present, 17 pounds, 3 ounces. Large, well-nourished infant. Lost 2 pounds, 10 ounces in six weeks with a severe attack of influenza. Is still taking citrate and gaining steadily.

CASE IV.—Michael A., six and one-quarter months, was on citrate two months. Weight, 15 pounds, 8 ounces; at present weighs 14 pounds, 10 ounces. Well-nourished breast baby. Lost 18 ounces the first two weeks. Is still taking citrate and is gaining steadily.

CASE V.—Rupert A., three months, was on citrate six weeks. Weight, 11 pounds, 2 ounces; after, 10 pounds, 14 ounces. On admission was put on a barley and milk mixture and lost 6 ounces

in three weeks. Seemed well and satisfied on the citrate, but had not gained when discharged.

GAINS.

CASE VI.—Agnes M., eleven and one-half months. Was on citrate five months. Weight before, 11 pounds; at present, 17 pounds, 6 ounces, or an average weekly gain of 5 ounces. Small, poorly-nourished infant who had not gained for two months. There was an initial loss on the citrate, followed by a steady and rapid gain.

CASE VII.—Helena F., eighteen months. Was on citrate three months. Weight before, 16 pounds, 8 ounces; at present, 19 pounds, 8 ounces. Child had not done well for several months. There was an initial loss, but she is now gaining rapidly. Vomiting, which was a distressing feature, has entirely ceased.

CASE VIII.—Bertha V., five months. Was on citrate two months. Weight before, 10 pounds; after, 11 pounds, 13 ounces. Was an habitual vomiter and lost on whey and cream mixtures. Vomiting ceased on citrate mixtures.

CASE IX.—Aileen D., two weeks. Was on citrate five weeks. Weight before, 7 pounds, 5 ounces; at present, 8 pounds, 7 ounces. Well-nourished breast infant put directly on citrate. Has gained steadily.

CASE X.—Joe S., five and one-half months. Was on citrate four months. Weight before, 9 pounds, 4 ounces; at present, 11 pounds, 15 ounces. Lost 17 ounces the first month on citrate mixtures, but is now gaining steadily.

CASE XI.—Dominic F., seven and one-half months. Was on citrate two and one-quarter months. Weight before, 11 pounds, 2 ounces; at present, 13 pounds, 12 ounces. Lost 2½ pounds in seven weeks on bariey and milk; then gained 1 pound on buttermilk. Made a steady gain on citrate until an attack of pneumonia. Is now gaining.

CASE XII.—Rosa R., three and one-half months. Was on citrate two and one-half months. Weight before, 7 pounds, 10 ounces; after, 8 pounds, 15 ounces. Lost 1 pound in seven weeks on barley and milk. Has gained irregularly on citrate.

CASE XIII.—Lewis B., seven weeks. Was on citrate two

and one-half months. Weight before, 6 pounds, 4 ounces; at present, 7 pounds, 3 ounces. Was put directly on citrate on admission and weight was stationary for several weeks. Is now slowly gaining.

CASE XIV.—Frank W., seven and one-half months. Was on citrate five months. Weight before, 8 pounds, 8 ounces; after, 10 pounds, 5 ounces. Poorly nourished infant. Had pneumonia and furunculosis. Did better on citrate than on whey mixtures and buttermilk.

CASE XV.—Irving P., eleven months. Was on citrate three months. Weight before, 11 pounds, 2 ounces; at present, 11 pounds, 5 ounces. Had influenza and pneumonia and lost 1½ pounds in six weeks. Is now gaining steadily.

CASE XVI.—Edith E., four and one-quarter months. Was on citrate four months. Weight before, 7 pounds; after, 8 pounds, 4 ounces. On whey mixtures gained 12 ounces in four months. Made a more rapid gain on citrate.

CASE XVII.—Maud S., ten months. Was on citrate three months. Weight before, 7 pounds, 13 ounces; after, 8 pounds, 2 ounces. An extreme case of malnutrition.

CASE XVIII.—Harold F., seven weeks. Was on citrate two months. Weight before, 5 pounds, 15 ounces; at present, 6 pounds, 10 ounces. Extreme case of malnutrition. Does not vomit as much on the citrate mixtures.

CASE XIX.—Douglas M., nine months. Was on citrate three and one-half months. Weight before, 11 pounds, 3 ounces; at present, 12 pounds, 8 ounces. Extreme case of malnutrition. Was steadily losing in weight until the citrate was added. Is now improving.

CASE XX.—Anthony E., two months. Was on citrate two months. Weight before, 6 pounds, 14 ounces; at present, 9 pounds, 3 ounces. Poorly nourished infant. Vomiting almost ceased after addition of citrate. The gain at first was very slow, but is now more rapid.

CASE XXI.—James S., eight and one-half months. Was on citrate two months. Weight before, 8 pounds, 14 ounces; after, 11 pounds, 1 ounce. Was an extreme case of malnutrition. Had been stationary for two months on whey. Initial loss on citrate, but afterwards a steady gain.

CASE XXII.—Mary M., four months. Was on citrate three months. Weight before, 15 pounds, 12 ounces; at present, 15 pounds, 12 ounces. Well nourished breast baby, put directly on citrate. Lost at first, but is now gaining.

The results were more satisfactory than anticipated. Five babies lost in weight, of which number one died. Sixteen gained and one was stationary. Two of the babies reported among the losing ones are still on the citrate and are now gaining steadily. In the one fatal case the feeding had nothing to do with the fatal result. The average gain was from ½ to 5 ounces a week. In several of the cases there was an initial loss on first taking the food, amounting to 17 ounces in Case X. This would be followed by a gradual and steady gain in most of the cases.

The control of vomiting in cases of chronic gastric catarrh was one of the striking effects of the citrate. Variot called attention to its benefit in this class of cases and Guyader[5] recommended before the Paris Société de Pédiatrie adding to each bottle a teaspoonful of a solution of 5 grams of citrate of soda to 300 cc. of water in cases of vomiting. Poynton found the citrate of special service in babies suffering from dyspepsia and vomiting. From the clinical results at St. Margaret's I can endorse these conclusions. It will only benefit those cases in which the gastric disturbance is caused by the proteids.

The effect upon the stools was variable. The reaction is usually acid, and undigested and offensive stools were found as before, but in no instance were tough casein curds found in the stools.

While the large majority of the babies were sickly and had been on other modifications, yet Cases III., IV., IX. and XIII. were well-nourished babies who had been breast-fed up to the time of admission. In Case IX. the gain has been steady. There was a loss at first in the other cases, but that was only temporary and the subsequent gain has been steady.

The preparation of the food mixtures was very simple. No attempt at a percentage modification was made. The milk was diluted in proportions varying from three parts of water and one of milk to three parts of milk and one of water. The water contained ordinary cane sugar in a 5 per cent. solution. The citrate of soda was kept in a solution so that each teaspoonful contained 10 grains. This was added to the food in the proportion of 1 grain to an ounce of milk. In some cases of habitual

vomiting the dose was increased to 3 grains to the ounce. The milk was neither pasteurized nor sterilized. There was no taste of the citrate even when added in a strength of 3 grains to the ounce and the babies took the food well.

The ease of preparation, the simplicity of modification, and the cheapness of the ingredients make this a valuable method in dispensary and institutional practice. In cases of proteid indigestion associated with vomiting and curds in the stools it has a distinct therapeutic value. The use of citrate of soda in infant feeding deserves a further clinical trial.

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DISCUSSION.

DR. GRAHAM.—I would like to ask whether in carrying out treatment in these cases any other medicine was used besides the citrate.

DR. SHAW.—Not a single one except in the very worst cases, where one or two saline enemas were used; nothing so far as the food was concerned.

DR. CHAPIN.—In adding anything to cow's milk we must keep two points in mind. First, the immediate effect of what is added; and, secondly, its remote effect. With reference to anything added in a chemical way we can easily determine the immediate effect. By a large addition of alkalies or of the substance just mentioned we can get immediate effects when it is desirable, but the more we tamper with milk the better our chances of eventually doing more harm than good. I have tried adding citrate of sodium to milk and it certainly makes it look different from pure milk, but I cannot help believing that this sort of research is in the wrong direction if we are trying to get something to use as a routine practice in the feeding of infants. Aside from the nutritional value of milk we must consider that it has an important developmental function and that milks and food principles are not interchangeable. Soluble proteids do not have the

same effect as insoluble proteids. This is an important law that we must bear in mind. It is an interesting study and we can tell pretty closely what this preparation is going to do with milk, but I think it is better to interfere with milk chemically as little as possible with reference to the good of the child.

DR. ABT.—I would like to ask the last speaker whether there is any difference between the citrate of soda and any other alkali.

DR. SHAW.—But this is not an alkali, it is a neutral salt.

DR. CHAPIN.—That is true, but the ultimate effect is the same.

DR. SHAW.—You can coagulate the milk if you use the proper dilutions as shown on this table. The idea is to relieve the stomach of some of its labor in digesting proteids. Nature meant the intestines to do part of the work and that is what this preparation facilitates.

DR. LA FÉTRA.—I would like to ask a question, too, as to the comparative value of citrate of soda in relation to other means used for throwing out stomach digestion. The alkalies have already been mentioned as well as lime-water and cereal gruels. In some cases of uncontrollable vomiting I have used citrate of soda, but without success. I wonder if Dr. Shaw has succeeded in cases where other things have failed or whether these are routine cases.

DR. KERLEY.—About two years ago, in making a study of comparative tests of retarded coagulation at the New York Infant Asylum, this subject was thoroughly gone into. Phosphate of soda was used in some cases, lime-water and carbonate of soda in others. While the formation of curd was prevented in the test tube we found that feeding the milk so treated was followed by loss of appetite, an insufficient quantity of the food being taken and the patients failed to thrive. My observation in infant-feeding bears out the point made by Dr. Chapin, which is that the less the milk is manipulated or treated chemically the better food it is for the child.

DR. SHAW.—In answer to Dr. La Fétra, I would say that this was a routine treatment for the purpose of trial.

In answer to Dr. Kerley, I want to repeat that citrate of soda does not act in the same way as the alkalies, in that it does not act by combination with the salts.

FAT INDIGESTION FROM A MOTHER'S MILK.

BY W. P. NORTHRUP, M.D.,

Professor of Diseases of Children in the New York University and Bellevue Hospital Medical College, Visiting Physician to the Presbyterian and Foundling Hospital of New York.

This is a tale of poisoning. It recounts a familiar story, how a mother's milk acted in a noxious manner upon her infant, with deleterious effect upon its health. You will recognize the words of the dictionary. It is a temptation to call it fat poisoning, but I fear someone will begin asking hard questions. I want further to leave a glow between the lines, "halation" I think the photographers name it, of even more important information. Among the many things to learn is what high specialization may lead to when once turned loose.

The infant under consideration was eight and a half weeks old when it came under my personal observation. Its symptoms then were those of disturbed bowels. The infant apparently was not very sick, was naturally small, its skin a little slack, as though the contained infant had shrunk a little, its demeanor indicative of a certain amount of general discomfort rather than colic or local pain. The important feature of its present condition was frequent and abnormal passages. It was the second child of young, healthy parents, born in uneventful labor, the elder child at present living and thriving. The mother loved to nurse her infant, and apparently had an abundance of milk. The infant took it, was five ounces heavier by careful weight after nursing than before; the child's passages were green and curdy, frequent and fluid. The provident parents had selected the best accoucheur in the city, who attended the mother for ten days, then passed the mother and infant to the care of an excellent family physician, both mother and child in good condition. Since the infant was on the breast, it was to be supposed that nature must be furnishing a milk in the mother's breast suited to the needs of the infant of her womb. Earthly prudence exercised its utmost in ensuring life's beginnings in the second born. With their first born the parents felt they had made mistakes, with the second they would make none. With the first born the physicians told the mother her milk

gave out because she did not eat enough, and did not do other things which they claimed they had advised. In any case, the little woman felt that this time she would leave nothing undone, this time she should not be blamable. She ate everything that came to her on the tray. Though her gorge rose at the sight and her stomach made strenuous involuntary contractions at the very savor of the approaching food, she engulfed it all. It may be remarked here that she was always indoors and took no exercise.

Between the third and fifth week the mother had tonsilitis, the temperature, reaching 104° F. Over the telephone my advice was asked, and I said, after learning that the attack was short and sharp with fever, that the infant might continue nursing so long as there was no appreciable disturbance. The fever speedily disappeared, the milk continued abundant and the infant thrived.

It was not long afterwards that the mother had a second attack of tonsilitis, in which it was believed the milk was disturbed. Of this attack I knew nothing until it was concluded that the baby's bowels were seriously deranged, the baby was losing sleep from colic, was losing weight. When I came to see the child and mother, I concluded that the mother looked too pale, too weak, too exhausted to continue nursing. In the eighth week the baby began to have frequent colic, several passages daily, brownish, frothy, containing milk curds, mucus in lumps; color olive green to brown, much gas.

The infant was receiving five ounces of breast milk from a devoted mother, in cool weather, its intervals of feeding and general care regulated by a trained nurse. One would think this was enough to ensure the thriving of the infant.

Acting on the thought that the disturbance was but slight and transient, that simple remedies would quickly correct the disorder, I cleansed the intestinal ways by a free dose of castor oil, directed that the mother should go out to drive in the open air, should sleep all night, feeding the baby twice during the twenty-four hours with weak modification of milk from the laboratory. She was to save her strength. She looked very pale and worried.

It seemed best to clean the baby's bowels again with oil, this time removing it from the breast entirely. It was fed on barley water for twenty-seven hours. During this time the baby was comfortable, though it acted hungry. The breast milk was tried again, colic, restlessness and gas returning at once. After that dilute feedings of Walker-Gordon milk with barley water, equal

quantity, were tried, and the baby at first thrived. (2 per cent. fat, 5 per cent. sugar, 0.75 per cent. proteids, one and one-half ounces, plus barley water one-half ounce.) The baby lost two ounces of weight. Digestion was for a time proceeding fairly well and the milk was increased to milk two parts, barley water one part, finally to full prescription. Directly mucus, colic, frothy passages again made it apparent that milk was out of the list of food for a time.

The mother looked very pale, bluish in the deep valleys under the eyes, was very nervous and worried about the baby. The extraordinary thing was that the breasts were full and the milk rich with cream. During this time the mother's milk was pumped and fully examined by Dr. E. E. Smith. Its fats were found to be 8.98 per cent., sugars 6.29 per cent., proteids 1.28 per cent. There was nothing extraordinary reported beyond an excess of fat. The particular symptom arising from such excess was diarrhea without vomiting. The passages were brownish, or between brownish and olive green, frothy, foul smelling, expelled with much gas, bubbles and seethings, with coarse fluttering sounds interspersed with loud detonations. The conspicuous feature was the wonderful gas-making capacity of the interior and contents of the intestines. There was also much mucus, tenacious masses, "wads." While this condition prevailed the baby cried all night with colic and slept none.

It was apparent that the baby could not yet take any milk. I gave castor oil once more to remove fermenting food and mucus. After castor oil the baby was kept exclusively on barley water for three days. When milk was resumed again colic reappeared promptly. The baby was always better on barley water. At the second attempt to complete the restoration of the intestine, cereal feeding was continued nine days. Barley water was given alternating with imperial granum and occasionally white of egg. Gradually, after nearly all mucus had disappeared, a single teaspoonful of "guaranteed" milk was added to a three ounce feeding of barley water, and gradually increased every second feeding, until four teaspoonfuls were taken in each. On the third day mucus reappeared, and the castor oil and barley water cycle was again traversed. This advantage had been gained, the infant could digest a little milk. From this beginning it went on to recovery. Irrigations were frequently resorted to, and were of great service. However, it was believed that the mucus was

formed partly in the ileum, and castor oil was needed to reach it.

Quite as much a part of treatment as anything else was the airing to which the patient was treated. The family had learned the good effects of fresh air treatment, and the little infant lay many hours in a bed at first in a distant part of the room, later in the air and sun close by the window, and it was the month of January.

One peculiarity which this infant manifested was a tendency to marked puffiness of hands and face on taking calomel in $^1/_{10}$ grain doses. The baby's urine was negative. Twice she manifested these symptoms in such degree that it was deemed wise to record a calomel idiosyncrasy against her for a few years at least.

I had great pleasure and help in having Dr. Jacobi see this infant with me. I thought no man could be better fitted to help feed a baby unable to take a trace of milk than he. He did it.

It is now six months from the acute stage, the infant is perfectly well and has been satisfactorily vaccinated. Has gained ten ounces during each of several weeks.

POINTS TO BE NOTED.

(1) This willing mother fell into misfortune through the conflicting purposes of too many excellent physicians. All these accidents would have been prevented if any one excellent person had had sole responsibility.

(2) Excess of fat produced in this case ileocolitis with these symptoms:—diarrhea (no vomiting), with frothy, brownish to olive green offensive passages, containing excessive amount of mucus in tenacious masses, colic, sleeplessness, loss of weight.

(3) Excess of fat was apparently produced by forced feeding, including a quart of milk daily, lack of exercise, indoor living, two attacks of tonsilitis.

(4) Fat was lowered by diet and driving in the open air, from 8.98 per cent. to 4.90 per cent.

(5) This infant had become extremely susceptible to the presence in her food of the least trace of milk and could not digest it.

(6) The first attack of tonsilitis apparently disturbed the baby's digestion somewhat. The second, with its consequent anemia, wholly upset it.

(7) Another time I should take the baby from the breast at once.

ACID AUTOINTOXICATION IN INFANCY AND CHILDHOOD.

BY JOHN LOVETT MORSE, A.M., M.D.,

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Vicarelli (1895) examined the urine of thirty perfectly healthy infants under six months for acetone. The distillate of the twenty-four hour amount was almost always positive by the iodoform test, but negative with sodium nitroprussid and oxalate of mercury. He concluded that the urine of healthy sucklings does not contain acetone. Baginsky (1888) found that acetone was present in the urine of healthy children under apparently entirely normal conditions, but only in very small quantities; while Langstein and Meyer (1905) found that the normal excretion in twenty-four hours never exceeded 1 cgm. Schrack (1889) says that the urine of children often contains small amounts of acetone from very insignificant causes. Brackett, Stone and Low, however, found acetone in only eleven of 300 surgical cases at the Boston Children's Hospital. In them it was associated with more or less severe symptoms. Langstein and Meyer (1905) found that when children from six to fourteen years of age received a meat diet without carbohydrates relatively more acetone was excreted than in adults. They also found that the principal part of the acetone was excreted through the lungs, and not through the urine as in adults. They always found diacetic and B-oxybutyric acid in considerable amounts. The organism of the child invariably reacted to the acidosis in the body by an increase in the excretion of ammonia. Camerer (1903) states that the nitrogen of ammonia in infancy is unexpectedly high.

The work of von Jaksch (1886) on acetonuria and diaceturia inspired Baginsky (1888) to study acetonuria in children. Schrack (1889) in the next year examined the urine of 400 children for acetone and diacetic acid. Their results and those of others, Bottazzi (1901), Freund (1903), Bernert (1904), Meyer (1904), in smaller series of cases, including one of 150 of my

own, tested for both acetone and diacetic acid by the sodium nitroprussid and ferric chlorid tests, apparently show that the acid bodies are present in the urine of infants and children under approximately the same conditions as in adults. They are often present in acute diseases accompanied by high fever (especially in the acute exanthemata, diphtheria and pneumonia), vary, as a rule, directly with the height of the fever and disappear with the fall of the temperature. I found but two positive reactions in 20 cases of pneumonia. In both the reactions occurred at the height of the fever, while the patient was taking sufficient food; and in both the reactions disappeared immediately after the fall of the temperature. In 7 negative cases the food was markedly insufficient in amount, although it contained a fair proportion of carbohydrates. These facts seem to show that the acid bodies were directly dependent upon the temperature. Meyer, however, claims, as the result of his observations in diphtheria, scarlet fever and measles, that acetonuria in the infectious diseases is independent of the sort or intensity of the infection and of the degree of fever, being due solely to carbohydrate inanition which is favored by the diminished power of carbohydrate metabolism in these diseases. In some instances, especially in typhoid, the acid bodies may persist, or first appear, during convalescence. They are then probably due to insufficient food or carbohydrates. In 15 cases of typhoid, in my series, 23 negative examinations were obtained in 13. The temperature was high in all these cases, and the food insufficient in amount in 4. Positive reactions were obtained in 2 cases at the beginning of convalescence; in one of these it persisted for eight days and only ceased when a sufficient amount of food was taken, although the food before was milk and barley-water. Acetone was present in 1 of 3 cases of bronchitis, and diacetic acid in 1 of 2 cases of pleurisy with effusion, while both were absent in 1 of empyema. Neither acetone nor diacetic acid were found in 2 cases of malaria, 3 of cerebrospinal meningitis, 2 of otitis media, 2 of pyelonephritis, 2 of rheumatism, 2 of cystitis and 1 each of acute nephritis and mastoid abscess. The food was markedly insufficient in several of these, although all took some milk. The acid bodies are said to be seldom present in tuberculosis of the lungs and intestine, but almost always in tubercular meningitis and miliary tuberculosis. They were absent, however, in 3 cases of tubercular

meningitis, all of which were taking almost no food, and in 1 of tuberculosis of the kidneys in my series.

The acid bodies in the urine are enormously increased in children during convulsions. Löbl found all the acetone bodies in a series of cases of tetany even when gastrointestinal symptoms were not marked. I did not find them in a case of tetany or in 1 of mild tetanus. They are, however, probably not the direct cause of the convulsions, because they are absent or present in only small traces in the urine of those diseases in which convulsions are most common, and because they may be present in large amounts without producing convulsions. They are not due to the lack of oxygen during convulsions, because lack of oxygen in conditions without fever does not cause them. The acid bodies and the convulsions are probably, therefore, independent of each other and merely manifestations of some common cause.

While the effect of fasting in the production of the acetone bodies has been pretty carefully worked out in adults, there are few or no data on the subject in infancy and but few in childhood. The effect of variations in diet has been but little studied at this age. Schrack (1889) never found the acetone bodies while a pure milk diet was being taken. They were found in a certain proportion of cases on a pure meat diet, and disappeared when an exclusively carbohydrate diet was substituted. Langstein and Meyer (1904) found the acetone bodies earlier and in relatively greater quantities in the urine of children from six to fourteen years of age who were fed on meat without carbohydrates than in adults under similar conditions. They assume that this is because the carbohydrate reserve in the liver and muscles is smaller than in adults. They call attention to the great danger of a fatty diet in children, when there is any disturbance of the carbohydrate metabolism. Czerny's and Keller's (1897) experiments on the excretion of ammonia in infants with gastrointestinal disorders point in the same direction. They found that the excretion of ammonia was much increased when creams low in proteids and sugar were given, while it was not increased when milks high in proteids and low in sugar or diluted skim milk with sugar added were given.

I have had no opportunity to study the effect of starvation for periods of more than forty-eight hours in infants and children. The food was withdrawn in all these cases purely for thera-

peutic reasons and not for experimental purposes. Neither acetone nor diacetic acid were found in the urine of infants under two years under these conditions, but diacetic acid was found in a child of two, and acetone in a boy of nine years, after twenty-four hours of starvation. It quickly disappeared in both when milk was given. No acetone bodies were found in fifteen infants that had taken markedly insufficient amounts of food for considerable periods and were not gaining or were losing in weight. The food, being some form of modified milk, always contained, however, relatively large amounts of carbohydrates. What little evidence there is goes to show, therefore, that the effects of starvation and variations in diet are essentially the same, although more marked, in childhood as in adult life. The data for infancy are too limited to warrant any definite conclusions, although they seem to suggest that the acetone bodies do not appear in the urine as the result of starvation as soon as in later life.

Both acetonuria and diaceturia had been previously noted in the acute gastrointestinal diseases of infancy by von Jaksch, Schrack and Baginsky. Attention was more particularly called to acid intoxication in these diseases, in 1897, by Czerny, Keller and Van den Bergh. Keller first noted that the excretion of ammonia was increased both relatively and absolutely. This has since been confirmed by Camerer (1903). Van den Bergh showed that this increase was due to an increase in the acids in the blood because it was diminished by the administration of alkalies. Czerny attributed the acid intoxication to abnormal products of decomposition of fats and proteids, but did not mention the acetone bodies specifically. The work and conclusions of Czerny and his school have been considerably discredited, however, in recent years. Vergely, in the same year, reported 20 cases in children, 5 of them in detail, in which he found the acetone bodies. In 1 case the course resembled that of typhoid fever. The acetone bodies were found as soon as they were sought, sometimes within a few hours after the onset. The acetone varied in amount from 10.5 to 12.5 grams per litre, and the B-oxybutyric acid from 6.8 to 8.5 grams per litre. The cessation of the symptoms and the disappearance of the acetone bodies from the urine were simultaneous. He divides the symptoms in these cases into two classes, nervous and gastric. The nervous symptoms are change of disposition, excitability, restlessness, ir-

ritability and insomnia; the gastric, anorexia, vomiting, thirst and constipation. There was no enlargement of the spleen, but slight enlargement of the liver. The temperature was sometimes elevated, sometimes not. It never lasted more than ten days. He concludes that these digestive troubles have a peculiar symptomatology, which is due to the presence of these acid bodies. He believes that the reason that the symptoms of acid intoxication are not more severe is because the eliminative organs are very active in childhood. Litten (1883) described 3 cases of a peculiar symptom complex in young children. A prodromal stage of a few days of mild symptoms of disturbed digestion was followed by marked nervous symptoms. The children became apathetic, whined continually, and could not be induced to move, play or take food. Sleep was interrupted and accompanied by groans and cries. An aromatic odor then appeared in the breath and drowsiness took the place of sleeplessness. One of the children had slight convulsive twitching. The type of respiration was not changed. With the appearance of the drowsiness diacetic acid was found in the urine. After two or three days the odor in the breath, the diacetic acid and the drowsiness disappeared together, recovery then being complete. These symptoms were altogether out of proportion to the severity of those in the digestive tract. He considers, however, that the primary cause is a disturbance of digestion. In a somewhat similar case reported by Krotkov, the diet for several days before had consisted almost entirely of fat pork. Similar cases in adults have been reported by Litten, Lorenz (1891) and Edsall (1902). Czerny (1897) has called attention to cardiac weakness and slowing of the respiration in infancy as the result of toxemia in the gastrointestinal canal, and says that the symptoms are like those of acid intoxication in animals. He does not mention the acetone bodies specifically.

All observers agree that the acetone bodies are rarely found in the urine in the chronic gastrointestinal disorders of infancy. My own experience leads me to believe that they are almost never present either in acute or chronic gastrointestinal diseases at this age. It is rather surprising that they are not because the limits of assimilation for sugar in infants with gastrointestinal diseases are much diminished, and the conditions for the development of an intermediary acidosis are thus present. In a number of these

cases, however, I have found sugar in the urine, but no acetone bodies.

The conditions seem to be markedly different in children, however. At this age the odor of acetone is not infrequently present in the breath, and the acetone bodies in the urine, not only in acute, but also in chronic disorders of the gastrointestinal tract. The odor of acetone in the breath, and the presence of acetone and diacetic acid in the urine, are especially common in the type of indigestion known as "chronic duodenal" not only during exacerbations but also in the intervals. Lorenz (1891) has already called attention to this fact in adults. I have found them pretty constantly in 2 cases during one and two years, respectively. This is not surprising, however, since the digestion of fat is especially interfered with in this condition. In a number of other children under my care, who are subject to disturbances of digestion, the odor of acetone in the breath is among the earliest, and is often the earliest, indication of an impending attack of indigestion. Examination of the urine at this time sometimes shows the acetone bodies, sometimes not. The peculiar odor in the breath is often associated with marked restlessness and irritability for some days before the development of other symptoms. Apparently impending attacks of indigestion can often be aborted or prevented in these cases by the use of bicarbonate of soda. In these cases the acid bodies appear when food sufficient, not only in quantity, but in the relative proportion of carbohydrates is being taken, and before the appearance of acute symptoms. They cannot, therefore, be attributed to starvation. These cases, while they resemble those in the group to be described later as recurrent vomiting, are more nearly like those described by Vergely. The symptoms of nervous irritability are marked and out of proportion to those of indigestion. They are sometimes accompanied by various forms of urticaria. The nervous symptoms cease with the disappearance of the acetone bodies.

There is no doubt that the acetone bodies are often present in small amounts in the urine in both acute and chronic gastrointestinal disorders in which there are no symptoms whatever of acid intoxication. In these cases they are certainly of no importance. There is no question, moreover, that in other cases there are marked peculiarities in the symptoms, especially in those referable to the nervous system, which coincide with the appear-

ance and disappearance of the acetone bodies. In these cases we are certainly justified clinically, in spite of the small amount of these bodies, in concluding that they play a certain part in the production of the symptoms. We are not justified, however, in concluding that acid intoxication is the only cause of the symptoms. In fact, it almost certainly is not. In certain cases, nevertheless, in which the symptoms resemble more closely those of diabetic coma, it probably is the most important cause.

There is great difference of opinion as to whether the acetone bodies are formed in the intestine in these cases, or in the body as the result of abnormal intracellular processes. The weight of evidence seems to show that they are not formed in the intestinal canal. It is probable, however, that they are indirectly dependent upon intestinal derangements which produce complex nutritional disturbances, which, in turn, result in the formation of the acetone bodies. The primary cause is almost certainly located in the intestine. Although the acetone bodies are secondary products, they do, in many cases, cause more or less characteristic symptoms, which not very infrequently dominate the symptom-complex. The demonstration of their presence is unquestionably, therefore, of value, both in diagnosis and in treatment.

Under the name of "vomiting in acetonemia," Marfan (1901) described a series of cases in children apparently identical in their symptomatology with those described in this country as "cyclic," "recurrent," or "persistent vomiting." The odor of acetone in the breath was always present at the beginning of the vomiting, and in 2 cases he detected it before the onset of the vomiting, showing that it was not due to inanition secondary to the vomiting. The odor of acetone either disappeared with the cessation of the vomiting or persisted for a day or two longer. Acetone was always, and diacetic acid sometimes, present in the urine. The amount of acetone in 3 cases was 0.38, 0.40 and 0.60 grams per litre, respectively. In some cases the acetonuria persisted for weeks after the cessation of the vomiting, although the children seemed in perfect health. In two instances several children in the same family were attacked simultaneously. He thinks that mild symptoms, such as loss of appetite and lassitude, may for a short time precede the onset of the vomiting. He thinks that these attacks have no connection with the kind of food, and that the exact cause is entirely unknown. He admits that the

acetonemia, by itself, has no precise significance, and considers it merely as an indication of a disturbance of the nutrition which may develop under various circumstances. He believes that the presence of acetonuria at the onset shows that it is not due to inanition, and that the disease does not originate in the stomach. He is not justified, of course, in assuming the presence of acetonemia because of the presence of acetone in the urine and breath. If the term, "Vomiting in association with the presence of the acetone bodies," is substituted for the term used by him, his argument seems sound, however. In the discussion of a somewhat similar case reported by Guinon (1902), Guinon, Merklen and Siderey all stated that in their opinions regulation of the diet and attention to the bowels will not prevent the occurrence of these attacks. Barbier also stated that in his experience the odor of acetone always preceded the onset of the attacks. In one of his cases the urine contained about ten times the normal amount of acetone per kilo. Edsall, in the next year (1903), reported several similar cases very briefly, including some of the cases reported more fully a little later by Peirson. In the case most carefully observed by Peirson, acetonuria and the odor of acetone in the breath always preceded the appearance of the vomiting; in 2 others, acetone and diacetic acid were found early in the attacks. He thinks that the onset of the vomiting is always preceded by other mild premonitory symptoms, as well as by the presence of acetone in the breath and urine. These symptoms are slight rise in temperature, loss of appetite, abnormal movements or slight nervous manifestations. He thinks that after these symptoms have appeared change in the diet will not prevent the attack. Both Edsall and Peirson succeeded in aborting or relieving the attacks by the free use of bicarbonate of soda.

In addition to seeing 2 of Peirson's cases, I have seen 2 others in which the odor of acetone in the breath, or acetonuria, preceded for twenty-four or forty-eight hours the onset of the vomiting. In several others I have found acetone or diacetic acid within the first twenty-four hours after the onset. In my cases also careful inquiry always revealed a prodromal stage of a day or two of slight indigestion, or nervous symptoms. Bicarbonate of soda relieved or aborted the attacks. Diet neither prevented nor relieved them. Some of these cases resemble, to a certain extent, those described by Vergely and others under the head of

acid intoxication in connection with gastroenteric diseases, the chief difference being in the mildness of the digestive symptoms before the onset of the vomiting. In others, however, it is very difficult to trace any connection with previous digestive disturbances or errors in diet. Moreover, regulation of the diet does not prevent their recurrence. In all, moreover, the severity of the symptoms is entirely out of proportion to the evidences of digestive disturbance. The presence of the signs of acid intoxication before the onset of the vomiting, their association with the vomiting, and the simultaneous subsidence of the two, certainly seem to show that they are intimately connected. The fact that the attacks of vomiting can be prevented, aborted or relieved by the administration of soda, the antidote for acid intoxication, shows that they must be due, in part, at least, to the acid intoxication. It is probable that the origin of the acid intoxication is not the same in all cases. In some it is probably due primarily to disturbances of metabolism, whose nature and cause are unknown, while in others it is probably primarily due to disturbances of digestion, which presumably induce secondary disturbances of metabolism.

It is undoubtedly true that the clinical picture of recurrent vomiting may be present without any evidence of acid intoxication and entirely independent of it. This fact does not prove, however, that in those cases in which the evidences of acid intoxication are present, it is not the cause, or one of the causes, of the attack. It merely shows that more than one pathological condition can produce the symptom-complex of recurrent vomiting.

It must be remembered, also, that the association of the evidences of acid intoxication and vomiting does not justify the diagnosis of recurrent vomiting to the exclusion of other conditions. This is well shown by a case reported by Leignen and Mirallié (1904), in which after a few days symptoms of tubercular meningitis developed, and were followed by death in three weeks.

Brackett, Stone and Low (1904) recently reported a series of 13 very curious cases occurring at the Boston Children's Hospital, on the surgical side, both before and after operation. They also quote a series of similar cases from literature. These cases all showed certain symptoms in common. These were: "Vomiting, associated with collapse; a very weak and rapid pulse; absence of fever; cyanosis, in the fatal cases, causing extreme dyspnea, apathy and stupor, alternating with periods of restless-

ness at first, but in the fatal cases, gradually deepening into coma and death; and the presence of acetone in the breath and urine." All these cases had extensive muscular atrophy as the result of paralysis or mechanical treatment. Autopsies were obtained in 4 cases. The only marked anatomical lesion made out was extreme fatty degeneration of the liver and muscles. They determined pretty conclusively that no single cause such as anesthesia, surgical shock, or change in diet, could have produced these symptoms. They conclude, however, that any one of these and other similar conditions may lead to changes in metabolism which, unless all the organs are sound and performing their functions normally, may produce serious or fatal complications. They attribute the symptoms to acid intoxication, and consider that the source of the acid bodies is the fatty degeneration of the muscles and liver. They finally conclude that the appearance of acetone in the urine, in quantities sufficient to give an ordinary clinical reaction, is to be regarded as an indication of serious, and possibly dangerous, disturbance of metabolism. Joslin estimated quantitatively the acetone excretion for twenty-four hours in 1 of these cases, a child of eight. The total excretion was 142 mgm., considerably more than the normal, but very much less than that found in diabetics, in whom it often reaches 100,000 mgm. This comparatively small amount of acetone would seem to cast some doubt on acid intoxication as the cause of the symptoms. In spite of this, however, the writer, who has seen a number of the cases, cannot help feeling that there is an intimate relation between the evidences of acid intoxication and the other symptoms in these cases, and that acid intoxication, whatever its cause, is the cause of the symptoms.

Conclusions.—The acetone bodies are not found in the urine of comparatively healthy infants and children by the ordinary clinical tests. They appear in their urine under approximately the same conditions as in adults. Certain disturbances of digestion associated with the presence of the acetone bodies in early life have peculiar symptomatologies. It is probable that the peculiar symptoms are due, in part at least, to acid intoxication. It is also probable that the acid intoxication is not primary but secondary. The connection of the symptom-complex seen in many cases of recurrent vomiting with acid intoxication is probably even closer than in the digestive disturbances just mentioned. In

these cases, also, the acid intoxication is presumably always secondary to some other abnormal condition, which may or not be digestive in origin. In any event, the etiology is obscure. In spite of the fact that the amount of the acetone bodies found in these conditions is relatively much smaller than those found in diabetes, the demonstration of their presence in connection with symptoms of gastrointestinal disturbance and the symptom-complex of recurrent vomiting, and probably also with other conditions, is of importance both in diagnosis and in treatment.

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DISCUSSION.

DR. EDSALL.—Dr. Morse has covered the general subject in a very complete way, and has so largely expressed my own views upon the subject that I shall do nothing more than mention a few details concerning some cases that I have seen since I directed attention, two years ago, to the relation between recurrent vomiting and acid intoxication. I feel in many ways still further convinced of the correctness of the views that I expressed at that time, and of the fact that some cases of recurrent vomiting can be controlled by treatment directed toward the acid intoxication. In my paper on the subject I mentioned a case that Dr. Griffith knows well, that of a child under the care of Dr. Sharpless, of West Chester, Pa. This child had such frequent and severe attacks that he was practically continuously incapacitated by them. I stated in my paper that the child was then improving, but had been treated too short a time to make the result at all clear. The further history has been that since the treatment with soda was started no attack has occurred, within about six months the child was able to attend school, and since this time he has had practically perfectly normal health. In another case, seen last fall, there were frequent mild attacks, and the general health was continually poor; that the child immediately improved in its general condition when put upon continuous doses of soda; there has been no attack since that time, and her general health is now practically entirely satisfactory. I have seen one further typical case, the child of a physician, in which the attacks were about as severe as they could be imagined, and their duration, severity and frequency had been increasing. Treatment in this case has not been as satisfactory; the child has improved, and in two years has had only three mild attacks, but it has not been possible to avoid attacks entirely. In all these instances large amounts of acetone and diacetic acid were present. In the first 2 cases I know that they were present at the very beginning of the attacks; in the last mentioned I have not been able to examine the urine at any time excepting when an attack had already lasted for several days. I have seen one other case that was referred to me as one of recurrent vomiting, but in which I think the diagnosis was questionable, since the child had a persistent and markedly dilated stomach. In this instance acetone and diacetic acid were not present at the time of the attacks, and whatever the nature of the case, treatment with soda did no good.

In my original paper I stated that I had little doubt that these attacks differ in their etiology, and I feel that this is almost unquestionably true. In some instances, however, I am convinced that acid intoxication is, from the standpoint of treatment, the most important factor, and in these cases persistent use of alkalies and careful control of the diet will ofttimes either entirely control the attacks, or will cause decided improvement. I have

been very forcibly impressed practically with one point that has been repeatedly demonstrated in the literature upon this subject, particularly in relation to the acid intoxication of diabetes mellitus; that is, the fact that acid intoxication, and the occurrence of symptoms therefrom, bears a close relation to a diet low in carbohydrates and high in fats. I think that careful investigation shows in a good many cases of recurrent vomiting that the child has had difficulty in digesting carbohydrates or is believed to have had such difficulty, and they have consequently been largely reduced or almost excluded, the deficiency having been made up to a considerable extent by increasing the fats. In this point, I think, lies one of the important elements in the perpetuation, and perhaps in the original production of the attacks, and also in the continuance of digestive disturbance of some other forms in children or older patients. In several cases of recurrent vomiting that I have seen, as well as in a number of other instances of gastrointestinal disturbance, I have found the children on a diet that contained very little carbohydrate, sometimes almost none, but a good deal of fat; and I have repeatedly seen, in various conditions, improvement in the symptoms and disappearance of acetone and diacetic acid from the urine, follow rapidly upon reduction of the fats, or their almost entire exclusion, and the use instead of considerable amounts of easily digested carbohydrates.

Some writers, in opposing the view that socalled acid intoxication *sui generis* produces the symptoms that occur in association with it, refer to the fact that the administration of alkalies will often not control the symptoms. This argument is entirely erroneous when it is applied to severe symptoms that are already present when the treatment is begun. Magnus-Levy in particular has emphasized the fact that in acid intoxication the acids probably unite not only with the alkalies of the fluids and tissues, but with the proteid tissues; it is well known that proteids readily form combinations with acids. The consequence of this must be an actual alteration in the tissues of the body, and consequently alkaline treatment, in order to be successful, must be begun early, before severe symptoms indicate that there have been extensive tissue-changes as a result of the circulation of the acids. To a certain degree the condition is comparable to tetanus and the use of tetanus antitoxin. The antitoxin is known to be a definite, direct antidote to the toxin, but it will not control the symptoms due to the tissue-changes produced by the toxin.

DR. KOPLIK.—Dr. Morse has laid much stress upon one fact which is borne out by my experience. In several cases of recurrent vomiting I have noticed the sweetish odor to the breath—the acetone odor. I recall very well one case, the child of a physician, a very good physiological chemist. The father examined carefully for the acetone bodies in the urine, but could not find

them. I have also noticed this odor to the breath in a case of Henoch's purpura and case of severe gastric disturbance.

DR. HOLT.—This is an extremely important subject, and one on which we need all the light we can get. I have not been so fortunate as to see all my cases relieved by the bicarbonate of soda; I have seen some improve, but not all. I have come to look upon the presence of these acetone bodies as practically diagnostic of the attacks. In several instances, where children had been having a number of socalled attacks of indigestion, which had not yielded to treatment, an examination of the urine set the diagnosis straight by revealing the presence of these bodies. There is another condition that has been brought to my mind two or three times which is often confused with recurrent vomiting, and that is cases of chronic appendicitis; under those circumstances the presence of these substances in the urine is a diagnostic point worthy of consideration.

It seems to me Dr. Morse has not laid enough stress upon the purely nervous influences in these cases. It is a marvellous condition that so many cases should have occurred in the children of physicians, possibly there is a nervous influence to account for it. I recall 1 case of a child who went to a kindergarten school for the first time and was very much interested in the proceedings, but had an attack that night. He was sent to the country and got well, but even now anything out of the ordinary will bring on an attack in that child. Some children will have attacks every month or so, and then without anything special being done, may appear well for a year. I think we should go slow about adopting the view that soda is a specific in these cases.

DR. ROTCH.—I have seen quite a large number of these cases, and have tried all forms of treatment, but at present know nothing satisfactory as to the cause or treatment. The soda treatment was seemingly successful in a few cases, but in many entirely failed. The attacks may stop for years and then come on again. In 1 case, which is now under observation, the attacks began when the girl was two or three years old, ceased for a number of years, and have begun again at the age of nineteen years, being now more severe than ever before.

DR. CRANDALL.—These cases certainly seem to be very contradictory, and it leads one to suppose that there must be more than a single cause. I have seen cases with the nervous element prominent, and others in which there was not any associated nervous condition. In a recent case the soda treatment gave brilliant results. The attacks were of the severe type, and the child vomited quantities of blood, but the soda controlled them.

As to the similarity to attacks of appendicitis, I know of 1

marked case that was operated upon, but no appendicial trouble was found. I have seen 2 other cases of supposed appendicitis that were relieved by the alkaline treatment and diet, so that I believe they were really cases of cyclical vomiting.

DR. TOWNSEND.—I had 1 case of recurrent vomiting who, after many attacks, had for the first time one associated with pain in the appendix region. A surgeon saw the case with me, and after the acute attack wore off did an operation, finding evidence of former attacks of appendicial inflammation, and since the operation the child has remained well.

DR. EDSALL.—I should like to say one word more, so that I may not be misinterpreted. From the remarks made by Dr. Holt and Dr. Rotch, I fear that I may be considered to hold that acid intoxication is definitely a disease. It is, of course, only a secondary condition, and it is always dependent upon some gross abnormality in metabolism or in the diet. I do consider, however, that in some instances it is, from the standpoint of treatment, the essential point to be held in mind.

DR. JACOBI.—In many of these cases acid intoxication is the first and prominent and persistent symptom in the clinical condition that may finally lead to acetonuria. Before a society like this, I am almost ashamed to repeat what I have had the good or bad luck of saying hundreds of times during the last forty years, the fact that the symptoms have been relieved by the substitution of cereal food—carbohydrates—in the place of fat. If there is one thing that will produce these symptoms it is an excess of cow's fat. Where food containing a smaller amount of cow's fat than the chemical requirements of the books would justify is used, the children never have those symptoms. I believe it is well known that I give very little fat as regular food; the proportion in the food I give is 2 per cent. or less. I have not the slightest doubt that what has been considered as a cure in these cases would be a good preventive if given as a regular thing.

DR. MORSE.—I fear that some of my statements have been misunderstood. I said that the symptom-complex of recurrent vomiting might be due to a variety of causes. In one variety we find the acetone bodies in the urine, and in these cases the symptoms are almost always relieved by the alkaline treatment. It seems fair to consider that in these cases the symptoms are due to acid intoxication.

In regard to the use of soda for a long time, it seems to me that if it is kept up for many months it may interfere with the nutrition of the child. I think it is wiser to watch such cases for premonitory symptoms, and give the soda then.

EYE SYMPTOMS OF INFANTILE SCURVY. A CASE OF INFANTILE SCURVY WITH EXTREME PROTRUSION OF THE RIGHT EYEBALL, SHOWN BY AUTOPSY TO BE DUE TO A LARGE RETROBULBAR HEMATOMA.

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The eye symptoms of infantile scurvy receive small notice in works on ophthalmology, and the writer has recently seen a consultation of eight oculists result in much difference of opinion as to the significance, cause, and treatment of proptosis in a scorbutic baby.

The history of the case is interesting, as it is one of the few where the cause of the protrusion of the eyeball was shown by autopsy to be an extensive subperiosteal hemorrhage of the orbital bones.

The patient was the offspring of healthy young parents, who had two older healthy children. It was born after a normal labor. It showed an intolerance of cow's milk, and was given cereal milk, upon which it throve for nine months, when it developed gradually pain, swelling and pseudoparalysis in both lower extremities. The loss of power was attributed to an obscure spinal injury by the family physician. Some weeks later, a slight protrusion of the left eyeball appeared, associated with blackened lids. Ten days later, a sudden protrusion of the right eyeball occurred, so extreme in degree that the closed lids left a large uncovered space on the cornea; both lids had a dark, bruised-like color, and were much swollen. Four days afterwards the baby became feverish and looked very ill.

The child was seen at 9 P.M., January 18th, by Dr. Lorenzo Burrows, who decided that the extreme proptosis of the right eye was not due to sarcoma, orbital cellulitis, or fracture of the skull, and referred the matter to Dr. Irving Snow as a medical case.

January 19th.—Examination by Dr. Irving Snow, 9 A.M. Child very apathetic; good development; slight rachitic rosary,

heart rapid and weak; nothing in chest or abdomen; temperature, 100°F.

Right eyeball protruding as in the most extreme case of exophthalmic goitre; cornea cloudy; upper and lower eyelids black and swollen, and do not cover the eyeball; no chemosis; eyeball freely movable; no discoloration of conjunctiva.

Moderate proptosis of left eyeball, with swollen, discolored lids; left cornea clear.

Gums slightly swollen and violet colored. No teeth.

Upper extremities, normal.

Legs below knee uniformly swollen and painful. Feet edematous. No purpuric spots or heat of surface.

The child thus presented a typical picture of infantile scurvy. The proptosis and swelling and pseudoparalysis of the legs were due to subperiosteal hemorrhage. Orange-juice, fresh milk and stimulants were given, but the temperature rose to 107.5°F. at 3:30 P.M. The child showed increasing prostration, and died at 11 P.M., after a stay of twenty-six hours in the hospital.

Autopsy.—An examination of only right eye permitted. A huge hematoma lay between the periosteum and bone of the orbit, filling the pyramidal space behind the eye and extending nearly around the entire orbit, stripping off nearly the whole orbital periosteum. No retinal hemorrhages. A smear from the clot showed large quantities of pus containing a bacillus like the influenza bacillus. Subperiosteal hemorrhage occurred in both orbits—in the left orbit, earlier and slighter in degree, and spontaneously ceasing. In the right orbit, an extensive, sudden hemorrhage took place, pushing forward the eyeball.

The rise of temperature in the last few hours of life was due to some terminal infection, probably proceeding from the infected retrobulbar hematoma.

The question of operative relief of the exophthalmos by removing the clot behind the eyeball was discussed, but was dismissed as unpractical on account of the moribund condition of the patient and the usual spontaneous absorption of the clot in a more favorable case.

Of the collective investigations of the American Pediatric Society[1] of 340 cases of scurvy, eye symptoms were mentioned in 49; a swelling mentioned in 9; in 18 protrusion of the eyeball; and in 22 both symptoms occurred. In the six autopsies reported

by the collective investigation, in none was an examination of the eye made. Heubner[2] reports 65 cases of infantile scurvy, orbit involved four times.

Regarding adult scurvy, Buzzard[3] (Reynolds' System) says, in some cases at an early period: "The integument around one or both orbits is puffed up into a bruise-colored swelling; the conjunctiva covering the sclerotic is turned and of a brilliant red color throughout; the cornea lies at the bottom of a circular trench or well. There is nothing inflammatory about the condition. It resembles a very violent ophthalmia in color, but with no pain or discharge."

Sudden exophthalmos in a baby should at once excite suspicions of scurvy. It may, in fact, be the first indication of the disease. The rapidity with which the proptosis occurs indicates a hemorrhagic lesion.

Barlow[4] (Grancher's System) says of infantile scurvy: "A remarkable symptom in one or both orbits is not rare. Suddenly, without premonitory sign or appreciable cause, we find a moderate exophthalmos, with the ocular globes turned downward. This symptom appears and augments for twenty-four hours. When it has reached its acme, one finds a deep ecchymosis and thickening of the superior lids, due to a hemorrhagic effusion. Both orbits may be affected, but successively and in an unequal manner. The proptosis may be only slight. There is no increase of intraocular tension or hemorrhage into the deeper parts of the eye. The eyeball is freely movable, not anchored by an inflammatory exudation, as in orbital cellulitis. The hemorrhage effusion into the eyelids affects the deeper parts; the conjunctiva of the lid and globe are only occasionally affected.

Similar cases of scorbutic proptosis have been collected, as follows:—

Barlow[5] (Keating's System) describes 2 cases of scorbutic proptosis. He says it is dependent on a bone lesion, an extravasation of blood between the orbital plate of the frontal and its subjacent periosteum, the extravasation pushing out the eyeball.

(1) Eight-months-old baby, brought on account of proptosis of one eyeball. Great tenderness in the limbs, and general cachexia. Since early infancy the baby has been rather tender in the legs. During the last week the tenderness became excessive; both upper eyelids became suddenly swollen three weeks

ago, the right upper lid purplish-red color, due to deep extravasation into its substance. No ecchymosis of palpebral or ocular conjunctiva. Slight proptosis of right eye. Cure after antiscorbutics.

(2) Ten-months-old baby, artificially fed. Legs tender at eighth month; at nine months the wrists became tender; there later developed ecchymosis in both eyelids; pseudoparalysis and slight proptosis of left eye.

Child died after an illness of three months. The autopsy showed subperiosteal hemorrhages. Eye not examined.

(3) Nicholai.[6] Nederlausch. Tijdschrift vor Geneekunde. Child nine months, brought to Nicholai for a sudden protrusion of the left eye. On both upper eyelids, there was a small greenish-yellow spot. Child otherwise well. Two days after Nicholai's first examination, a neuroparalytic keratitis developed, and Nicholai found in the gums the characteristic changes of scurvy. A few days later a retrobulbar hemorrhage occurred in the right eye. On antiseptic washes and antiscorbutics the keratitis and ocular protrusion disappeared.

(4) Ed. Meyer.[7] Seven-months-old child, previously well, attacked with edematous swelling and pain in left ankle; left-sided exophthalmos. Death with increasing anemia. Autopsy.—Rachitis, anemia, hemorrhagic pachymeningitis, hemorrhagic periostitis of the left orbit and tibia, fatty myocarditis, hemorrhages into heart, lungs and kidney.

(5) Ashby and Wright.[8] Girl, seven months old, fed on dried milk and maltose-food for seven months. Throve and looked like a prize baby. Bad cough for two to three weeks. Two weeks before examination, the left eyeball became suddenly prominent, and protruded so much that the nurse thought it would drop out. The proptosis was attributed to a slight blow on the eye the baby received from a comforter. The eye had been prominent for two weeks, during which time the baby had several bad fainting attacks. The baby was large and fat and pale; temperature 101°F.; drowsy, but readily aroused. The eyeball was exposed in part; the lids when closed did not meet; lids not puffy or ecchymosed; ribs beaded; no teeth, gums normal. Urine stained the diaper brown. Rapid improvement on fresh milk and orange-juice.

It is interesting to note that so acute and experienced an observer as Dr. Nansen[9] looks on adult scurvy as a disease not due

to the absence of a certain element in the food, but to the presence of ptomaines in badly preserved salt beef or other preserved foods. He believes that with sterilized foods and properly given, there would be no scurvy, and that orange and lime juice are unnecessary. Both Dr. Nansen and Dr. Caillé believe that scurvy in adults and children is due to ptomaine poisoning.

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TEMPERATURE, PULSE AND RESPIRATION RELATIONSHIPS IN INFANCY AND CHILDHOOD.*

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At the suggestion of Professor J. P. Crozer Griffith, I have undertaken a study of the relation the pulse and the respiration bear to the temperature and to each other in febrile conditions in children, omitting from consideration those diseases in which the pulse or respiration is known to be unusually slow or rapid.

It is a matter of common observation that the respiration and the pulse are very irregular in children, and are more markedly so the younger the child. Some writers even maintain that it is practically impossible to determine any normal relationship for a given age. Yet physicians recognize that in certain diseases the pulse or the respiration or both may be unusually slow or unusually rapid, a recognition which implies that at the different ages there does exist some range of figures within which pulse and respiration should fall for each degree of temperature. Moreover, every pediatrist is able from experience to estimate that a certain pulse rate is too high, although none can easily tell just what is meant by this. In medical literature little, if any, information can be found on the subject.

I have therefore attempted to discover what figures at the various ages in infancy and childhood represent the ordinary ratios that the increase in the pulse-rate and the increase in the respiration-rate should bear to the degree of increase in the temperature, and that the pulse-rate and the respiration-rate should bear to each other.

In adults the ratios are fairly definite, an increase of ten pulse beats and two respirations and a half for each degree of increase in the temperature and a pulse-respiration ratio of 4:1, as given by Handford,[10] Griffith,[8] Aitken,[1] and others, being accepted generally. These figures, of course, are not found in cases of pyrexia when the heart, lungs, or meninges are involved, in typhoid fever and scarlet fever, and in some other affections (Donne,[5] Handford,[10] etc.); even in diseases where these ratios are the rule, the

* Presented before the Society at the request of Dr. Griffith.

greatest variations from them may undoubtedly occur (Smoler,[17] Billet,[8] Liebermeister,[12] Monti,[14] etc.).

Before taking up the study of the ratios that exist when the temperature is elevated I have sought to determine what are the ordinary pulse- and respiration-rates at the different ages in infancy and childhood when the temperature is normal.

THE NORMAL AFEBRILE PULSE- AND RESPIRATION-RATES AT DIFFERENT AGES IN CHILDREN.

A number of pediatrists have given what they regard as the normal pulse- and respiration-rates to be expected in healthy children of different ages; their figures follow:

ASHBY AND WRIGHT.[2]	Newborn.	Early months.	End of 1st yr.	3-4 yrs.	5 yrs.	8 yrs.	12 yrs.	15 yrs.
Respiration...	44	35-40	28	25	..	..	..	20
Pulse...	130-133	120-140	110	..	100	90	80	..

COMBY.[4]	Birth.	1st months.	3-8 months.	1-2 years.	6 years.	12 years.
Respiration...	35	..	..	27-25	22	20
Pulse.....	138-140	130-135	120-125	115-120	..	..

GRIFFITH.[9]	Birth.	1st mo.	1-6 mos.	6 mos.	1 yr.	1-2 yrs.	2-4 yrs.	6-10 yrs.	10-14 yrs.
Pulse.....	130-150	120-140	130		120	110-120	90-110	90-100	80-90
Respiration	30-50, av. 40		25 35, average		30	28	25	20-25	

MONEY.[13]	1st weeks.	1st year.	1-4 years.	5 years.
Pulse	150-120	100-120	100	90
Respiration	30-50	25-35	..	..

MONTI.[14]	1st day-2 mos.	2 months-1 year.	2 years.	3-5 years.	6-10 years.	10-15 years.
Pulse.....	140-120	130-100	120-90	110-72	104-64	80-60

PARRY AND HODGE.[15]	Under 6 months.	6-12 months.	12-18 months.	18-30 months.
Pulse..... ..	125	124	115.5	111.8
Respiration.....	44.8	34.8	35.5	29.8
Cases.....	27	2	4	7
Number of observations	90	11	24	37

ROTCH.[16]	Early weeks.	Until 2d year.	2-3 years.	3-5 years.	5-8 years.
Pulse.....	120-140	110	100	..	90
Respiration..	45	15 40		25	..

SQUIRE.[18]	1st few months.	2d to 7th year.
Pulse.....	120	100
Respiration	40	24

STARR.[17]	Birth-2 mos.	2-6 mos.	6-12 mos.	1-3 yrs.	3-5 yrs.	5-10 yrs.	10-12 yrs.
Pulse.....	160-130	130-120	120-110	110-100	100-90	90-80	80-70
Respiration..	44	35			23		

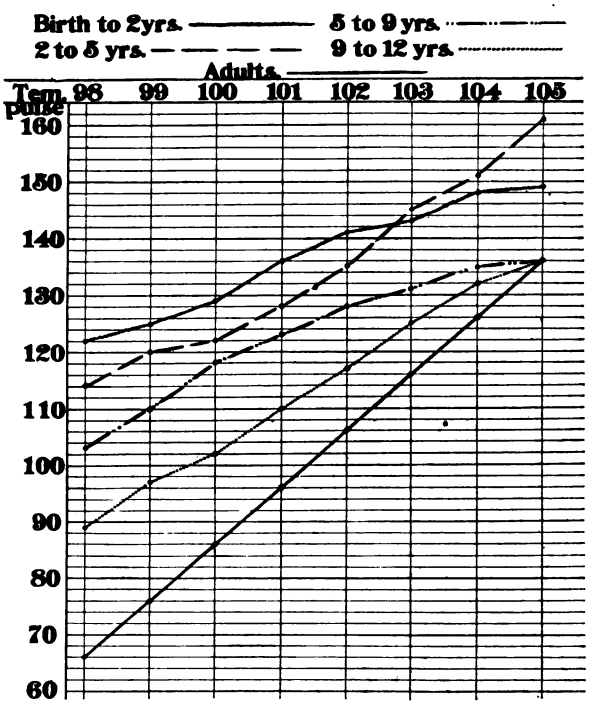
TAYLOR AND WELLS.[21]	Newborn.	2d year.	7-14 years.
Pulse.....	120-140	100-115	80-90

THOMPSON.[22]	Birth.	6-12 mos.	1st year.	2-4 years.	2-6 years.	7 or 8 years.	11-14 years.
lse.....	120-140	115-105	..	..	105-90	..	85-75
espiration	32-50	..	25-35	25	Higher than adults.		

The figures given by most of these writers appear arbitrary and schematic instead of based upon scientific investigations; Parry and Hodge are the only ones to record the number of cases and observations used in determining the averages.

In recording the results of my observations I have divided infancy and childhood into four periods: (1st) birth to the

PULSE CURVES AT DIFFERENT AGES WITH THE DIFFERENT DEGREES OF TEMPERATURE.



end of the second year, *i. e.*, infancy; (2d) two years to the end of the fifth year, *i. e.*, early childhood; (3) five years to the end of the ninth year; and (4th) nine years to the end of the twelfth year. The average pulse-rate and respiration-rate I obtained for the normal temperature (98°) in each period and the number of cases and observations from which those average rates were calculated are given in the table below.

It should be borne in mind that the observations were made on

children who were sick or convalescent, and that no particular effort was made to examine the children when asleep or exceptionally quiet. Nevertheless, inasmuch as I excluded all cases in which the disease could be expected to materially influence the pulse and respiration, my figures may be regarded as fairly average ones.

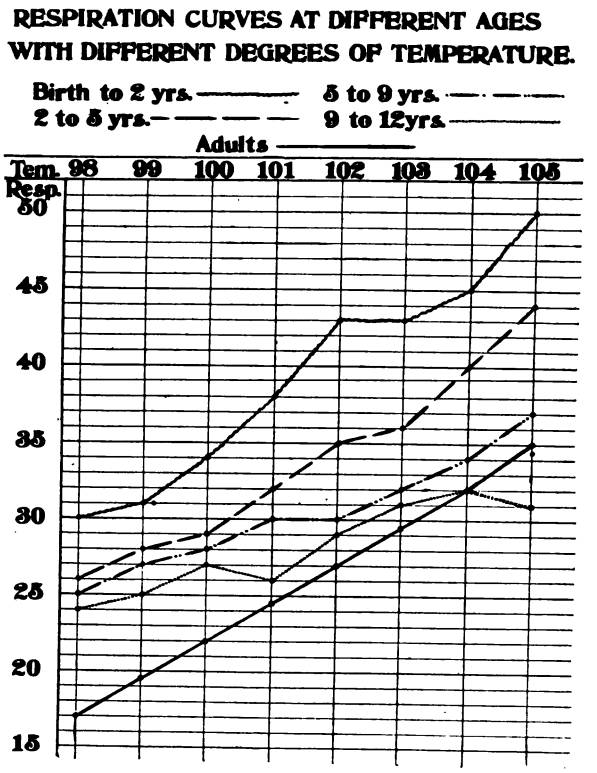


TABLE I.

THE AVERAGE PULSE AND RESPIRATION-RATES IN CHILDREN WITH NORMAL TEMPERATURE.

	Birth to 2 years.	2 to 5 years.	5 to 9 years.	9 to 12 years.
Pulse-rate	122	114	103	89
Respiration-rate	30	26	25	24
Number of cases	44	40	29	16
Number of observations	370	335	257	151

The statistics of the normal pulse-rate and respiration-rate given by the different observers cannot be easily combined owing to the different groupings of the ages. My figures, being based on a fairly large number of cases and observations, may be therefore taken as probably representing the average.

These figures show that although the pulse-rate and the respiration-rate are more rapid the younger the child, they do not decrease uniformly with advancing years. The respiration-rate, on the one hand, decreases four in passing from the first to the second period, and then one in passing to each of the remaining periods. The pulse-rate, on the other hand, decreases more uniformly, being eight less in the period from two to five years than in the one immediately preceding, eleven less in the period from five to nine than in the one before, and fourteen less in the fourth period than in the third, the amount of decrease for each period being always greater by three than that for the period immediately preceding. This difference between the pulse-rate and the respiration-rate as regards the amount of decrease produces a variation in the normal pulse-respiration ratio, which is approximately 4:1 for the first period, 4½:1 for the second, 4:1 for the third, and 3¼:1 for the fourth. These ratios, as has been stated, are for afebrile states.

THE PULSE- AND RESPIRATION-RATES IN FEBRILE CONDITIONS IN INFANCY AND CHILDHOOD.

In endeavoring to ascertain the average pulse-rate and respiration-rate in febrile conditions I examined more than 7,000 case-histories, but discarded all those in which there was any evidence of cardiac, pulmonary or meningeal involvement or of malnutrition, as well as all cases of typhoid fever, scarlet fever and rickets. This was done because the pulse or the respirations or both are well known to be abnormally slow or rapid in these conditions. There was then left a total of 162 cases, a number by no means large enough to admit of positive assertions, but still of sufficient size to furnish some guide.

As already stated, the years of childhood were divided into four periods, namely: Birth to the end of the second year, two years to the end of the fifth year, five years to the end of the ninth year, and nine years to the end of the twelfth year. I endeavored, when possible, to obtain in each case at least ten records of the pulse and of the respirations for each degree of temperature, with

which I included the fifth above and the fifth below. From these I calculated the average rates for that case. I also tried, when possible, to secure in each period at least 10 cases for every degree of temperature. The average pulse and respiration for each period was obtained by combining the average rates of all the cases in that period. Below is a table giving the number of cases and observations I was able to obtain for each degree of temperature in each period.

TABLE II.

NUMBER OF CASES AND OBSERVATIONS.

	Birth to 2 years.		2 to 5 years.		5 to 9 years.		9 to 12 years.	
	Cases.	Observations.	Cases.	Observations.	Cases.	Observations.	Cases.	Observations.
98°	44	370	40	335	29	257	16	151
99°	44	487	26	275	33	353	17	170
100°	40	390	34	334	35	257	17	189
101°	36	181	36	169	33	258	18	88
102°	33	122	29	149	36	251	15	63
103°	22	64	27	110	25	117	8	57
104°	22	51	19	103	23	55	7	22
105°	5	11	9	21	6	12	2	5

THE AMOUNT OF INCREASE IN THE PULSE-RATE PER DEGREE OF TEMPERATURE AT DIFFERENT AGES.

The relationship between the temperature and the pulse in febrile cases has been very little studied. Jordan[11] even asserts that no such relationship exists; but he draws this conclusion after studying cases of scarlatina, enteric fever, empyema and tuberculous meningitis, conditions in which the ordinary relation of the temperature, pulse and respirations is commonly disturbed.

In children under four years of age Gerhardt and Seifert[7] find the pulse-temperature (Centigrade) ratio of increase to be 10:1 (Fahrenheit 5.5:1) with mild febrile reaction, and still greater with higher fever; in children over four years of age they believe the ratio to be 10:1 (Fahrenheit 5.5:1) with high, but less with lower temperatures.

Monti,[14] in a table apparently schematic and not based upon actual observations, gives the pulse-temperature (Centigrade) ratio of increase as 12:1 (Fahrenheit 6.6:1) for children under two years of age, and as 10:1 (Fahrenheit 5.5:1) for children over that age.

Handford[10] gives no figures; but states that in children over ten years of age the pulse is more rapid than in adults.

Smith [19] also makes the mere statement that febrile and inflammatory diseases produce a greater acceleration of the pulse in early life than in maturity.

The following table gives the average pulse I obtained for each degree of temperature at different ages, with the pulse-rate in the adult added for the purpose of comparison.

TABLE III.

AVERAGE PULSE-RATES IN FEBRILE CONDITIONS IN CHILDREN.

	Birth to 2 years.	2 to 5 years.	5 to 9 years.	9 to 12 years.	Adults.
98°	122	114	103	89	66
99°	125	120	110	97	76
100°	129	122	119	102	86
101°	136	128	123	110	96
102°	141	135	128	117	106
103°	143	145	131	125	116
104°	148	151	135	133	126
105°	149	161	136	136	136

Despite the great variations which undoubtedly exist, it is nevertheless possible from the observations recorded to make an approximate estimate of the average increase in the pulse with each degree of increase in the temperature. This may be stated as four in the period from birth to two years, five in the period from five to seven years, and seven in the periods from two to five years and from seven to twelve years, as compared with an increase of ten in adult life. Why the second period should exhibit a greater degree of increase than the third I am unable to explain. Such a condition may be shown by further studies to be not constantly present, although, on the other hand, it may constitute an actual exception to the rule one would expect, namely, a progressive increase in the ratio.

The general trend of my observations nevertheless appears undoubtedly to bring out the surprising and interesting fact that, although for the same degree of temperature the *actual number* of pulse-beats tends to be greater the younger the individual, the *amount of increase* in the pulse tends to be greater the older the individual. It would thus appear as though fever obliterates the difference between the pulses of different ages; as though there is almost a common pulse

rate for high temperatures which all cases, irrespective of age tend to reach. Infants, starting with the highest afebrile pulse, naturally, as the temperature rises, have a lesser number of pulse-beats to cover in order to reach this figure; consequently they exhibit the least ratio of increase, only 4:1. Adults, starting out with the lowest afebrile pulse-rate, have the greatest number of pulse-beats to cover; hence their pulse-temperature ratio of increase is the greatest, being 10:1. These conclusions, it will be noted, are in direct contradiction to the view quite commonly accepted, which is expressed, for instance, in the quotation from Monti.

There are, of course, numerous cases where the pulse-rate in early life is higher than my averages show. Various accidental causes, however, may account for this; such cases can hardly be considered as fairly representative ones. The circumstance, moreover, that appears in my table and in the graphic chart, of the pulse being greater in the second period than in the first when the temperature is over 103°, may be accidental or may represent an actual exception to the tendency stated above.

THE AMOUNT OF INCREASE IN THE RESPIRATION-RATE PER DEGREE OF TEMPERATURE AT DIFFERENT AGES.

The only writer to refer to the ratio of increase in the respirations is Handford,[10] who says that in children over ten the respirations do not seem to be so much accelerated as the pulse.

The average respiration-rates I obtained for each degree of temperature at the different ages are given in the following table, in which, as before, I have inserted for comparison the respiration-rate in the adult.

TABLE IV.

AVERAGE RESPIRATION-RATES IN FEBRILE CONDITIONS IN INFANCY AND CHILDHOOD.

	Birth to 2 years.	2 to 5 years.	5 to 9 years.	9 to 12 years.	Adult.
98°	30	26	25	24	17
99°	31	28	27	25	19.5
100°	34	29	28	27	22
101°	38	32	30	26	24.5
102°	43	35	30	29	27
103°	43	36	32	31	29.5
104°	45	40	34	32	32
105°	50	44	37	31	34.5

As in the case of the pulse, it is possible to estimate the average amount of increase in the respiration-rate with each degree of increase in the temperature. This may be stated as 3 in the period from birth to two years, 2½ in the period from two to five years, 2 in the period from five to nine years, and 1½ in the period from nine to twelve years, a difference of half a respiration in each period. The respiration-rate resembles the pulse-rate also, in that for the same degree of temperature the actual number of respirations tends to be greater the younger the child.

Yet for the same degree of temperature the amount of increase in the respirations tends also on the whole to be greater the younger the child, a condition just the opposite to that of the pulse. Thus, while the temperature is rising from 98° to 105° the respiration-rate is increased by 20 in babies under two years, but only by 7 in children over nine years of age.

Consequently, as regards the respirations, fever does not seem to obliterate the difference between ages, there being not the slightest tendency for the respiration-rates of the different ages to approximate as the temperature rises; in fact, rather the reverse seems true.

THE RATIO OF THE PULSE-RATE TO THE RESPIRATION-RATE IN FEBRILE CONDITIONS IN INFANCY AND CHILDHOOD.

The pulse-respiration ratio (*i.e.*, the ratio of the actual pulse-rate to the respiration-rate, not the ratio of their amounts of increase) has not been studied by many writers. Thompson[28] fixes it at 3½:1 or 4:1 in health and believes that any great disturbance of these ratios is of clinical significance. Squire [18] states that notwithstanding the readiness with which the breathing and the pulse vary in infancy under the slightest exciting causes, they maintain a relation to each other which is characteristic of the early period of infancy and which differs from the ordinary ratio of childhood and adult life. He finds the actual ratio of the pulse to the breathing only 3:1 in early infancy, and even less, and when this is steadying down to the more mature ratio of 4:1 he believes that any causes that quicken the rate also increase the ratio.

The pulse-respiration ratio according to my figures is practically 4:1, except in infants under two years of age with a temperature above 100°, and in children from two to nine years with a temperature above 104°. In these cases it is practically 3.5:1, except in infants with a temperature of 105°, in whom it is 3:1. The exact figures are given in the following table.

TABLE V.

PULSE-RESPIRATION RATIOS IN INFANCY AND CHILDHOOD.

	Birth to 2 years.	2 to 5 years.	5 to 9 years.	9 to 12 years.	Adult.
98°	4.1:1	4.4:1	4:1	3.7:1	4:1
99°	4:1	4.3:1	4:1	3.9:1	4:1
100°	3.8:1	4.2:1	4.2:1	3.8:1	4:1
101°	3.6:1	4:1	4.1:1	4.2:1	4:1
102°	3.4:1	3.9:1	4.3:1	4:1	4:1
103°	3.3:1	4:1	4.1:1	4:1	4:1
104°	3.3:1	3.8:1	4:1	4.2:1	4:1
105°	3:1	3.6:1	3.7:1	4.4:1	4:1

From this table it would appear that in children under five years of age the pulse-respiration ratio is less, the higher the temperature; but that in children over five years the ratio is influenced by the temperature very little or not at all. A general conclusion may be drawn that the pulse-respiration ratio is 4:1 in children of all ages and at all temperatures, except in infants with any amount of fever and in children from two to nine years of age with hyperpyrexia, in whom the ratio becomes 3:1 or 3.5:1.

In closing I desire to express my thanks and appreciation to Professor J. P. Crozer Griffith for placing his clinical material at my disposal and for rendering me invaluable assistance and encouragement in the preparation of this paper.

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DISCUSSION.

DR. HOLT.—I suppose we are all somewhat surprised by these results. As regards the pulse, it is a question whether a larger number of investigations would bear out these conclusions. I am speaking particularly, of course, of the first two years of life. Personally, I should like to see a larger number of cases studied before we consider this question settled.

Another source of error should be considered. Observations

of this kind cannot be made properly unless made with reference to the particular question studied. If ordinary history records are used for analysis, and the pulse and temperatures are taken under different circumstances, sometimes asleep and sometimes awake, one will get varying and unreliable results. The nurses made many of these examinations, too, and you are all familiar with the unreliability of nurses' records as regards pulse rate.

DR. JACOBI.—My remarks would be similar to those made by Dr. Holt. I would not, from my experience, trust a nurse for examinations of this sort. I would not, from my experience, trust most senior or junior medical assistants, but I would trust most, but most only, house physicians to make such observations. Furthermore, every one of those observations should be made during sleep; anything else is unreliable.

DR. HAND.—Whatever exceptions we may take to the way this data was collected I think we may consider the conclusions as safe, as regards the first two groups of figures particularly. Whenever I find a pulse rate of 180 in a sick child I begin to feel anxious about the outcome of that case. I have seen a few cases of 180 persistent pulse recover, but they are very rare.

The only exception I would make to the chart is that perhaps the figures for the normal pulse rate of the young child are rather high.

DR. GRIFFITH.—I am glad to see this skepticism, because I had it myself at first. We are prone to be influenced by our preconceived notions of what things *should* be. When I first saw these figures, I said: "There must be something wrong. Look them over again." Dr. Cohen did this, as I requested, but found them correct. When you come to think of it, you will perceive the reasonableness of what Dr. Hand has just said. The infant's pulse *cannot* increase 10 beats per degree of temperature, as the adult's does; otherwise, it would, with every hyperpyrexia, be dangerously rapid.

Now, regarding the criticisms which have been made, I have this to answer. First, inspection of the figures contained in Dr. Cohen's paper will show that they are for most temperatures and ages quite large enough to allow of conclusions being drawn with propriety. Second, it is objected that the estimations of the pulse were made by nurses and, therefore, cannot be depended upon. This seems to me a rather scathing arraignment of the ability of a nurse to do what she is trained to do. She may be unable to judge properly of pulse tension and strength, and their significance, but surely she should be able to count the number of beats per minute. In any case, Dr. Cohen's paper presents to you only the methods which each one of you is following every day. You enter the sick room or the hospital ward and examine the tem-

perature record taken by the *nurse,* and the rates of pulse and respiration taken by the *nurse.* We publish records of cases based upon these. I only ask you to compare these records with the records presented you this evening. The objection which one speaker has raised, that he has often found the child's pulse 30 or 40 beats higher than that which the nurse has recorded, is very readily explicable. The nurse's record was probably correct. The child is more used to her presence, and the approach of the physician would naturally send up the pulse rate. Third, it was objected that the pulse rate ought to be taken during sleep. This is probably true. As is well known, the rate is probably 20 or 30 beats per minute less than when awake. I am glad the speaker mentioned it, because the record during sleep would make the pulse rates on Dr. Cohen's chart even lower than they are at present, and make them illustrate even more the point which I am making, viz., that with high fever all pulse rates at all ages tend to be the same.

NOTES OF A CASE OF ACUTE LEUKEMIA.

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AND

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Although numerous cases of acute leukemia in childhood have been reported, and the subject itself has recently been discussed at length by members of this Society, the affection is still sufficiently rare and obscure to demand that a careful record be made of each case. The notes of the following case, which appears to us to possess features of special interest arising from its abrupt onset and extremely rapid course, we think desirable to place on the records of this Society:

A. M., a lad of Jewish descent, aged fifteen, born in Chicago, but living in Montreal for the past three years, was admitted to the Montreal General Hospital on June 4, 1904. On May 29th, seven days previously, he was awakened in the morning by a profuse epistaxis, which continued all that day and part of the next. There had been no injury and his parents could assign no cause. The day previously, the 28th, he had been at school and was stated by his parents to have been in good health and his skin of a normal color. He was bright, played with his friends as usual, and went to bed, as far as they knew, in perfect health. On two or three previous occasions he had suffered from bleeding at the nose, but each of these attacks had been promptly stopped with simple remedies. Otherwise he had always been healthy, except for an attack of mumps. His diet, so far as could be learned, was wholesome. The portion of the city in which he lived is one of the most healthy, being comparatively close to the mountain. The family history is an unusually good one with no tendency to hemophilia in any of its members.

The epistaxis which had continued all through the day of the 29th, and part of the 30th, had recurred on the 31st and had been checked by plugging the nares with gauze. On June 1st he began to bleed from the mouth, and a continuous oozing from the mucous membrane of gums and of both cheeks had persisted until the time of his admission. Ecchymotic spots had also appeared on the lower extremities. On June 2nd vomiting and diarrhea had set in; the vomitus contained blood, but there had been no melena or hematuria. Headache was complained of and vertigo, with shortness of breath on any attempt at movement.

When seen in the hospital he was in a very restless state, tossing from side to side on the bed; mentally, he was dull, but could be roused to answer questions. The rectal temperature was 101°F., pulse 132 and respirations 32. Examination showed him to be a fairly well nourished boy; the skin, mucous membranes and conjunctivæ showed a marked degree of anemia; petechiæ, varying from 1 to 5 mm. in diameter, were scattered over the trunk, and more numerously on the extremities, especially on the legs below the knees where several large ecchymoses were also to be seen. There was no edema. The nares were plugged with gauze. Petechiæ were visible on the buccal surface of the cheeks and there was a continual oozing of blood from the edges of both gums. The tonsils were slightly enlarged and the breath was foul-smelling. The joints were normal. Marked tenderness was elicited by pressure over the sternum, but over no other bone. There was a moderate discrete enlargement of the superficial lymph glands, the anterior and posterior cervical, the axillary, the epitrochlear and the inguinal were easily palpable, but tenderness in them was not elicited by pressure. The respirations were slightly increased in frequency, but examination revealed no abnormal conditions in the lungs. Transverse cardiac dulness measured 7 cm.; there was a slight systolic murmur at base. Blood pressure registered 120 mm. by the Riva Rocci apparatus.

The abdomen was distended, especially in the upper zone, and the outline of the enlarged spleen was distinctly visible. On palpation the lower margin of the liver was easily felt 6 cm. below the costal margin in the nipple line and tenderness was elicited on pressure over it. The spleen was distinctly palpable and its resisting border could be felt 6.5 cm. from the median line and 6 cm. below the costal margin.

Hearing appeared to be normal. Vision in the right eye was blurred. An examination of the field of vision was impossible on account of the patient's condition and unfortunately no examination of the fundi was made.

The urine was somewhat turbid, its specific gravity 1.012; it was acid in reaction and contained neither albumin nor sugar. Under the microscope only a few leukocytes were to be seen, but no blood cells, no casts, and no uric acid crystals.

An examination of the blood made immediately on admission by Dr. Patterson, to whom we are also indebted for many of the notes, showed the erythrocytes, 1,620,000; leukocytes, 34,000; hemoglobin, 35 per cent.; clotting was normal.

The lad's condition became rapidly worse. Numerous loose movements with no evident signs of blood in them occurred. He vomited all nourishment taken. The petechiæ and ecchymoses increased in number and in size; vision was lost in the left eye on the morning of the 6th. Death took place the same evening.

A second blood count made on the morning of the 6th showed:

Erythrocytes	1,390,000
Leukocytes	91,600
Large Lymphocytes	45.5 per cent.
Small Lymphocytes	34.5 " "
Polymorphonuclears	12 " "
Myelocytes	3.0 " "
Transitionals	4.2 " "
Eosinophiles	.8 " "

The small lymphocytes were quite normal, the nuclei of the larger ones were perhaps more granular than usual. In a count of 500, three nucleated cells were seen. A culture from blood taken at the same time was carefully made by Dr. Gillies, but proved negative as the tubes showed no growth after forty-eight hours.

AUTOPSY.—The autopsy was performed by Dr. Gillies sixteen hours after death. The body was that of a well nourished boy; postmortem rigidity was present, also postmortem lividity over the dependent parts of the trunk. The skin was of a pale lemon tint showing over the abdomen, chest, legs and arms numerous petechiæ, and over the lower extremities, large purpuric patches.

The external genitals were normal, the pupils equal and the gums were covered with clotted blood.

On opening the abdomen the peritoneum was seen to be glistening; the liver was seen exposed for one hand's breadth below ensiform cartilage, and the spleen reached the costal border at the level of the ninth left costal cartilage. On removing the sternum the thymus gland was seen to be enlarged.

The pleuræ, lungs and pericardium were normal. The *heart* was slightly enlarged, and its muscle pale. Lying beneath the visceral pericardium, on the anterior surface of the ventricles, close to the septum, were seen numerous fairly large ecchymoses. The *spleen* was large, its weight 430 grams; its capsule was slightly wrinkled; its consistency increased. On section, the organ cut firmly and presented a somewhat mottled color, varying from a dark brownish-red to a dark yellow; the Malpighian bodies showed distinctly on the dark background. The *right kidney* was of normal size; its weight 100 grams; its color pale; the venæ stellatæ were prominent. The capsule peeled off readily from the surface of the organ, showing several whitish areas, not raised above the level of the surrounding kidney substance, and of varying size. On section, the cortex and medulla were seen to be of a pale yellowish color, and scattered over the cut surface were irregular whitish masses, similar to those seen on the outer surface of the organ. The pelvis was normal. The *left kidney* presented the same general appearance as the right, but the whitish masses were fewer in number. The *suprarenals* were normal. The *liver* was slightly enlarged, its color brownish-yellow, its weight 1,330 grams, its capsule smooth, its edges sharp and its consistence normal. On section the color was yellowish and the lobules somewhat indistinct; the parenchyma was friable. Scattered over the cut surface were seen yellowish-white masses, which were less distinct and smaller than those in the kidneys.

The stomach was somewhat dilated and showed submucous ecchymoses. In the duodenum, immediately beyond the pylorus, was seen a localized infiltration of the walls resembling a tumor 3 cm. in breadth. The surface was elevated and the mucous membrane eroded. The lymph follicles throughout the small intestine showed hyperplasia and the Peyer's patches were much enlarged. The colon was normal. The pancreas was normal. Behind the

pancreas was a large mass of swollen lymph nodes, of firm consistence, one node being nearly as large as a small lemon. The edge of the mass extended as far as the site of the eroded area in the duodenum. Some of the nodes in the mass were of a reddish hue, while others were whitish and firm. The mesenteric lymph nodes were swollen and soft, as also were the inguinal, the axillary and the cervical nodes. The rectum was normal. The testicles were normal. The thymus was enlarged, soft and gelatinous on section; the thyroid colloid. The larynx and trachea were normal. The esophagus was normal. The abdominal aorta was smooth and elastic.

MICROSCOPICAL APPEARANCES.—The duodenum, in a specimen taken from the tumor-like swelling immediately beyond the pylorus, showed an infiltration of large round mononuclear cells into the mucosa and submucosa between Brunner's glands. The cells were not granular and among them might be seen a few small mononuclear cells resembling small lymphocytes. The normal tissues of the gut in this section seemed little disturbed by the presence of these cells. A section at the level of the tumor, however, presented a different microscopical picture. Here all signs of the normal structures were lost, except in the neighborhood of the outer edge of the tumor where a few strands of muscular fibres were seen passing in among masses of mononuclear cells. The cells presented no definite structural arrangement; in places a reticulum could be made out about each cell, but in other places the reticulum was lost and the cells were massed together and held in place by a coarser mesh. The blood vessels were poorly developed. The microscopical appearance resembled that of a lympho-sarcoma. The mucosa was completely absent from the surface of the tumor.

The capsule of the spleen was seen to be somewhat thickened, the parenchyma everywhere showed marked hyperemia, the sinuses being distended with blood cells. The Malpighian bodies were enlarged and showed a general infiltration with mononuclear cells of a uniform type, in which the nucleus was relatively large and the protoplasm clear.

The liver cells in general stained well and the nuclei were clear and distinct, but in many there were evidences of fatty change. In many places about the portal vessels were to be seen collections of mononuclear cells; masses of these cells were also

scattered irregularly throughout the lobules, leading in some places to the complete disappearance of the liver cells. It was possible to trace in places the various stages from that of slight compression of the liver cell to that of complete replacement, the stage depending upon the number of newly formed mononuclear cells. In some instances atrophying liver cells could be seen within a mass of these round mononuclear cells.

The kidney showed the cells of the glomeruli distinct, with clear staining nuclei. In the convoluted tubules the cells were somewhat swollen and the nuclei indistinct, but in other parts the cells stained clearly. Throughout the section masses of well stained mononuclear cells were to be seen forming the whitish masses visible to the eye on section. In some places these compressed the normal tissues about which they lay, and in other places replaced them completely. Where the new-formed cells were few in number no change could be detected in the pre-existing structures.

The mesenteric, retroperitoneal, cervical and axillary lymph nodes all presented the same microscopical appearance. Among the ordinary cells of the lymph nodes were to be seen numerous large mononuclear, non-granular cells. In some of the retroperitoneal nodes polynuclear cells were also present. The structure of the lymph nodes was preserved.

The bone marrow showed myelocytes, both neutrophile and eosinophile; also red blood corpuscles, both nucleated and non-nucleated. The large mononuclear lymphocyte predominated all other white cells, and in comparison to it all the other white cells were relatively few in number.

This case, both in its clinical symptoms and its anatomical findings, is, we think, to be regarded as a typical example of acute lymphocytic leukemia, as recognized at present. It began, as many of these cases do, abruptly with hemorrhagic manifestations. Close cross-questioning of both parents and the lad himself revealed no indication of ill-health in the boy previous to the initial epistaxis, except that in answer to a leading question the lad said that after running he had perhaps felt a little out of breath for the past three weeks. It had not, however, interfered with his games. Counting from this initial epistaxis the course of the disease was just ten and a half days. The case in this respect is therefore especially interesting, for so far as we have

been able to investigate the subject, the cases which have ended fatally in twelve days, or less, may be counted on the fingers of one hand. Litten[1] would seem to hold the record, his case lasting only four and a half days. Nobel[2] reports one the duration of which was ten days. Greiwe's[3] case lasted eleven days, and those of Englisch[4] and Obrastzow[5] lasted twelve. McCrae[6] also reports a case in a lad of fifteen years, which ran its course in about twelve days.

The question is an interesting one as to what the exact condition of the lymph nodes and blood in this lad was immediately preceding the hemorrhage. While there was nothing in his appearance or in his behavior to attract the attention of his parents, who were rather above the average of their class in intelligence, we recognize that in a certain number of cases a latent stage commonly known as the aleukemic stage exists, and it is possible that a careful examination at that time might have revealed some deviation from the normal.

Dr. Thos. McCrae,[6] in speaking of 5 cases that occurred in the Johns Hopkins Hospital, gives as the average of their blood count on admission: Hemoglobin, 35.4 per cent., red cells, 1,822,000, and leukocytes, 104,000. In the case we have just reported the hemoglobin was 35 per cent., the red cells 1,620,000, and the leukocytes 34,000 (1—48); but in thirty-six hours the leukocytes had risen to 91,000 (1—15); at the same time the large lymphocytes were greatly increased, the polymorphonuclears were diminished and myelocytes were present.

Dr. McCrae considers that in acute leukemia we have to recognize in addition to what may be called the specific leukemic features, grave changes in the red cells themselves, suggesting that in acute leukemia, in addition to the changes in the white cell count, we have to deal with a very rapid and severe anemia of the primary type. Nevertheless, he admits that in the majority of cases, and especially in rapid cases, there is very slight variation in the red cells in size, shape and staining. In the present case there was almost no poikilocytosis and no polychromatophilia. A careful recount showed a few nucleated red cells with no mast cells; the character of the staining was extremely uniform.

According to Pinkus,[7] of Berlin, children are attacked with relative frequency. This must hold true of the second rather than of the first decade. Of 45 cases recorded by Theodor,[8] 6 oc-

curred in the first decade and 5 between the ages of ten and fifteen years; and in 56 cases collected by Fussell,[9] Jopson and Taylor, 9 were in the first decade and 5 between ten and fifteen years. In the 5 cases reported by McCrae, one was aged three and one fifteen. Nevertheless, Churchill,[10] in his paper read before the Society last year, was only able to collect 29 cases under ten years of age reported in medical literature.

On the relative frequency of acute leukemia statistics, beyond indicating its comparative infrequency, are not very definite. McCrae states that in 17,100 admissions into the medical wards of the Johns Hopkins Hospital, there were thirty-seven with leukemia, and of these only five were acute. In Montreal, out of 9,930 medical admissions into the Royal Victoria Hospital, there were 21 cases of leukemia, of which 5 were regarded as acute, and in 10,350 admissions to the medical wards of the Montreal General Hospital, there were 8 cases of leukemia, of which only one was acute.

Acute leukemia in childhood differs in few respects from the disease as met with in adult life. Hutchison,[11] following Guinon and Joly, describes three types of the disease. In the first there is a profound anemia with general glandular enlargement, but hemorrhages appear only in the latter stages of the disease. In the second type the hemorrhagic diathesis is manifested from the outset, petechiæ appear early and the case may assume the aspects of an infective purpura. In the third type, lesions of the buccal cavity are the most striking features.

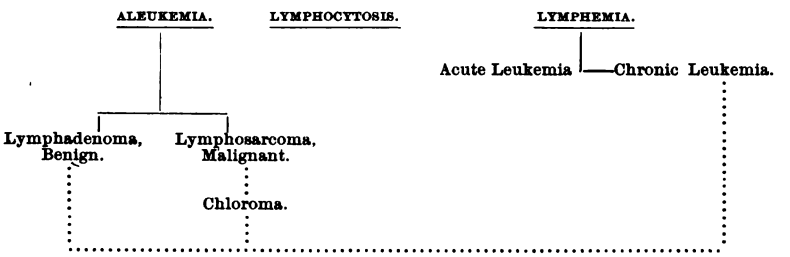
While the bone marrow is more or less affected in every case, cases are reported in which no splenic or glandular enlargement is present. Kelly[12] has called attention to the fact that while the diagnosis is to be made from the blood examination alone, a single enumeration is not always sufficient. In one of Kelly's cases the leukocytic count showed only 5,200 leukocytes, yet the differential count showed over 90 per cent. of lymphocytes.

Between acute leukemia and chronic leukemia there is no definite dividing line, and although certain authorities have regarded the two affections as different entities, the clinical symptoms and blood examinations present no essential point of difference between the rapidly malignant and the more indolent forms of the disease.

That there may be a close relationship between leukemia and

pseudo-leukemia would appear very probable. In an interesting paper published by Dr. Martin[13] in 1896, attention was called to this fact, and to the tendency to fall back from the present somewhat sharp classification of these diseases and to return to the simple grouping of Trousseau, who included under the single term *adenie* all lymphoid hyperplasias.

In recent lectures Turck,[14] under the term lymphomatosis, included three sub-divisions according to the following scheme, the dotted lines indicating that a connection exists between the affections thus linked together.



In a recent paper he states that he would include under the term lymphomatosis all true hyperplastic tumor formation of lymphoid tissue due to formative disturbances. Thus into one group with this appelation he places all cases of lymphatic leukemia, acute leukemia, pseudo-leukemia, lymphosarcoma, lymphosarcomatosis, chloroma, malignant lymphoma and, perhaps, also, splenic anemia. The fundamental basis of the group he regards as an increased stimulation of growth in the lymphatic system. The etiology is unknown. The difference in the manifestations depends upon two factors:

1. The degree of the increased growth and its local behavior.

2. The presence or absence of a washing out of cells, the result of this growth, into the peripheral blood stream. In one subgroup all the new cells go to the formation of tumor masses and the blood picture is unchanged. The blood finding is alymphemic. In a second and larger sub-group, besides the cells making up the tumor masses, there is an increased washing out into the blood of the cells representing the product of this increased growth, the lmphocytes.

Turck does not deny that the bone marrow may be involved, and even admits the possibility of this being occasionally the sole site of the disease, but he does deny the absolute dependence of the lymphemia upon the blood marrow changes in all cases.

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A CASE OF ACUTE ALEUKEMIC LEUKEMIA IN A BOY TWO AND ONE-HALF YEARS OLD.

BY ALFRED HAND, JR., M.D.,

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The notes of this case are as follows: R. V., male, two and one-half years old, was admitted to the Drexel Children's Hospital March 11, 1905, with a negative family history, both parents and all the other children being alive and well. The patient had been breast-fed for the first seven months of life; he had had whooping-cough when three months old and measles at one year; otherwise he had been in robust health until the beginning of the present illness seven weeks previously. At that time his mother was confined to bed for a few days and he received little attention, living in a cold room most of the time; after this he seemed to have something like the grippe, which was followed six weeks ago with swelling of the feet and lower eyelids. He had neither headache nor vomiting, but he was constipated and passed a diminished amount of urine, and he became steadily paler.

Condition on Admission.—General development, good; marked anemia; lower eyelids so puffy that the eyes are almost closed; considerable hemorrhage under the skin and also under the ocular and palpebral conjunctiva; numerous petechiæ on forehead and on cutaneous surface of lips; numerous submucous hemorrhagic spots, 2 mm. in diameter on dorsal and under surfaces of tongue and on buccal mucous membrane; on the abdomen, near the right anterior superior iliac spine is a black-and-blue spot 2.5 cm. in diameter; smaller spots are sprinkled over the abdomen and legs, but not so profusely as on the forehead. The mind was clear, there was no retraction of the neck, no exaggeration of the knee jerk, no ankle clonus, and Kernig's and Babinski's signs were negative. The breath-sounds were normal everywhere over the lungs. The heart-action was rapid, but the sounds were clear throughout the course of the case. The liver was palpable, 6 cm. below the costal border and the spleen 3 cm. Only one urinalysis is recorded, on the third day after admission, neither albumin nor

sugar being present, the sediment consisting of epithelial cells and leukocvtes. The urine was frequently passed in the bed, but it was possible to collect and measure the following daily amounts: 520 cc., 150 cc., 370 cc., 470 cc., 290 cc., 330 cc., so that the kidneys seemed to be acting well, with a minimum of 17 ounces on some days, the rest being lost.

The blood count showed red blood corpuscles, 1,390,000; leukocytes, 6,300; hemoglobin, 23 per cent. There was considerable difficulty in stopping the flow of blood from the puncture of the finger, but pressure finally succeeded after about an hour. The result of the blood count led us to fall back on the diagnosis of purpura hemorrhagica, but as the lymphatic glands, axillary and inguinal, became palpable in a few days, and with the result of the differential count of the leukocytes we were led to regard the case as one of acute leukemia of the lymphatic type, with the leukemic manifestation held in abeyance by some unknown condition. A. O. J. Kelly has reported from the laboratory of the German Hospital such a case, in which syphilis was present, and he thinks it possible that the absence of increase in the leukocytes might have resulted from the associated lues. In this case we are at a loss to know what could have been the deterring factor. The differential counts were made both by the resident physicians and myself with striking similar results, a count of 700 leukocytes giving:

Polymorphonuclears	8.85	per	cent.
Large mononuclears.....	4.73	"	"
Lymphocytes	85.00	"	"
Myelocytes	1.42	"	"

The stained red corpuscles showed microcytes, macrocytes, poikilocytes and normoblasts.

Four days after admission nose bleed occurred and was followed in a few hours by vomiting of clotted blood. Subsequently the bowel-movements were dark brown in color, but the vomiting after that did not show blood, so it is possible that the intestinal tract was not the seat of hemorrhages.

The temperature ranged between 99.5° and 101° for three days after admission and then remained close to the 101° line for three days more, rising on the seventh day to 105.4°, falling the next morning to 101°, when death occurred from exhaustion. It is to be regretted that an autopsy was not permitted.

As the case stands it is, of course, incomplete and the naming of it is largely a matter of speculation. It was evidently an infection whose manifestations were localized mainly in the blood and lymphatics, and as acute lymphatic leukemia is so considered, and as this form does not necessarily have as high a leukocyte count as the other forms, it seems more reasonable to class the case here than elsewhere. I prefer to consider it as such rather than purpura hemorrhagica, because the bleeding was not the most prominent symptom and because of the differential count. Malignant endocarditis is not absolutely excluded in the absence of an autopsy, but the heart-sounds never showed even the softest murmur, there were no signs of embolism and the kidneys were unaffected.

By a curious coincidence the issue of the Journal of the American Medical Association for March 18, 1905, the day the patient died, refers to a case reported by Dr. Geo. Blumer, of acute Hodgkin's disease, the description of which is strikingly like the above, the author remarking that perhaps it would be better to consider it as acute aleukemic leukemia.

DISCUSSION OF THE PAPERS OF DRS. BLACKADER AND GILLIES AND OF DR. HAND.

DR. HOLT.—There is nothing upon which we need greater light than these acute blood conditions; the difficulty of knowing where many of these cases are to be placed is very great. I saw a case two months ago that in my experience was unique and it is suggested by these two reports. A little girl, eight years of age, seemed quite well and attended school on Wednesday; later in the afternoon she amused herself by jumping rope. On Thursday morning she was taken with vomiting and a temperature that quickly reached 104°F. On Friday, the vomiting and fever continued and there took place a free hemorrhage in the bladder; the family physician thought she passed about a pint of blood; the quantity was surely large, but it was mixed with urine. By Saturday there was present a condition of intense toxemia with steadily high temperature and great prostration. On Sunday, the first blood count was made, and the red cells had fallen to 1,500,000, the leukocytes were 133,000, and the hemoglobin was only 20 per cent. I saw the child on Monday and her general appearance strongly suggested that of typhoid in the third week. There was no jaundice though she had that pale, yellowish green color. The nervous symptoms were very pronounced, the tongue

was dry and there were sordes on the lips. The prostration was intense and there was a soft systolic murmur which we thought was hemic. Greatly to our surprise the child began to improve on Tuesday, and by Thursday the nervous symptoms disappeared, the red cells increased to 3,000,000, and in ten days the hemoglobin had arisen to 60 per cent.

The picture is one I have never seen before, and the most striking feature was the outcome, for it looked as if the child was in a hopeless condition. What the nature of the case was I am unable to say. The blood showed no evidences of malaria or typhoid. The changes were those of an intense secondary anemia with marked leukocytosis.

DR. KOPLIK.—There is one point brought forward by the readers of both papers, and that is the importance of not coming to a conclusion on the strength of one leukocyte count. That point was brought out by Dr. Osler in his excellent paper on leukemia. He showed that in adults the blood might return to a normal standard in this disease and remain there for some time. This was beautifully illustrated in a case of acute leukemia in a child thirteen months of age that was under my care. When I first saw the child, all the symptoms of acute leukemia were present as we are accustomed to see it in the adult, and I took the child into the Mt. Sinai Hospital and studied it for some time. With a red count of 1,500,000, the leukocytes were 96 per cent. of the white cells. I made repeated counts and, strange to say, for a week in the course of this leukemia the blood count returned to a perfectly normal standard with the normal relative relation of the lymphocytes to the polynuclear leukocytes. When the resident physicians were thinking that some mistake had been made in the diagnosis and I was preaching Osler to them the change occurred, the child gradually increased its lymphocytes, running up to 90,000 and before death, which took place a week or so afterwards, developed a marked lymphocytosis. The case shows the necessity for frequent counts and the fact that one leukocyte count means very little.

Regarding the duration of some of acute leukemia, I saw a case the other day in which I had made the diagnosis six months ago and predicted a fatal issue at that time. I received a message a few weeks later that the child had improved. I saw it again recently, after an attack of diphtheria; the picture of leukemia had become marked again and the child died because of a recrudescence of the blood disease after the attack of infectious disease.

DR. EDSALL.—I reported a case at the last meeting of the Association of American Physicians that presented some features that were of particular importance in connection with children. The patient was an adult who had a rapidly progressing acute leukemia. When I first saw him the most striking point was the

close resemblance to diphtheria. He had become gravely ill within a few days and had fever, nasal obstruction, with a watery blood-stained discharge from the nose, patches of pseudomembrane in the nose, difficulty in swallowing and a high grade of laryngeal stenosis. When first seen there was no pseudomembrane on the tonsils, but twenty-four hours later both tonsils were covered with necrotic pseudomembrane, and at that time I think it would have been impossible without culture to have excluded diphtheria. This point in the diagnosis of acute leukemia is, so far as I know, not clearly emphasized in the literature. It is, however, of importance because necrosis of the tonsils and pseudomembrane formation is quite common in acute leukemia.

Another point is perhaps worth mentioning. In most cases of acute leukemia there is a tremendous increase of metabolism, and particularly a remarkable increase in the destruction of nuclein-containing tissues. It has been suggested by several men, first of all, I think, by Dr. Alonzo Taylor, that it might be possible in the earlier stages of acute leukemia or in cases in which the diagnosis remains persistently obscure, to get help from a determination of the uric acid of the urine. The uric acid output has been found in many instances to be enormously increased, and has reached even to the point of 8 grams a day. Dr. Taylor and I did some work along this line in connection with children a few years ago, but without any satisfactory results; and it now seems to be that the method is not promising in relation to diagnosis because, apparently, the great increase in uric acid excretion occurs only late in the disease. Still it might perhaps be of value in those cases that run throughout a course that is more or less obscure. It would, however, be perhaps quite as useful to make an estimation of the urea as of the uric acid, because the urea is also enormously increased. Clinical urea estimations are, of course, very inexact, but in this instance only gross changes would be of much importance, and probably even an inexact method would give fairly useful results if any help in diagnosis were to be gained by this means.

DR. MORSE.—It seems to me that the diagnosis of acute leukemia is hardly justified in Dr. Hand's case, and that the condition he describes can be explained in another way.

DR. ABT.—A case came under my observation recently that emphasizes at least one difficulty in making the diagnosis of these cases of lymphatic leukemia. A boy, thirteen years of age, who had been ill during his infancy and early childhood, suffering from rickets, laryngismus stridulus and malnutrition. When he was about six years old he met with a severe injury by being run over, both knee-joints were opened and the surrounding tissues crushed. His present illness began in March of this year. He complained of pain in his extremities, he was tired, became breath-

less on effort, his appetite was poor, and he lost some in weight. In the meantime the anemia became progressively more marked, and the spleen was continually decreasing in size. When I first saw him he was very anemic, much reduced in weight, the hemoglobin per cent. (Fleischl) was about 50 per cent. There were about 2,000,000 red corpuscles and 21,000 leukocytes. The differential count showed a preponderance of the lymphocytes, which were present to the extent of 70 per cent. But at this time he had a very severe tonsillitis and pharyngitis. The spleen was large, extending as far down as the level of the umbilicus. At this time it was thought that possibly he was suffering from a lymphatic leukemia, but in a few days with the diminution of the throat symptoms, the number of leukocytes fell so that now there were but 4,000 present, and this condition of leukopenia has continued. In other words, I believe that the little patient was sick with a splenic anemia, that the picture of this disease was changed by an intercurrent pharyngitis and tonsillitis which caused the disease to resemble for a few days an acute lymphatic leukemia.

DR. BLACKADER.—I think we have all noted the fact which has been illustrated in such an interesting way by Dr. Koplik's case, namely, the great recuperative power manifested occasionally by the elements of the blood; we would suppose, *a priori,* that the child would have this power of recuperation to a greater degree than the adult.

DR. HAND.—I have only to emphasize the remarks made by Dr. Holt, which reminds me of Dr. Osler's remark concerning the erythema group, that we need a Linnæus to bring us out of the dark.

A STUDY OF LEUKOCYTE COUNTS IN FIFTY CASES OF BRONCHOPNEUMONIA, LOBAR PNEUMONIA AND EMPYEMA IN CHILDREN.*

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In children, as in adults, the term leukocytosis is applied to a more or less transient increase in the number of the white blood corpuscles. In certain malconditions of childhood there occurs also a constant and essential over-production of the leukocytes, as in the pseudoleukemia of v. Jaksch. Though various theories have been offered to explain the temporary increase in the white cells occurring in acute diseases such as pneumonia, in truth little is known as yet about the exact pathogenesis of leukocytosis. In a general way, however, we have learned from the studies of Ehrlich and Wasserman that chemotactic influences must play an important rôle in the causation of the process.

A view that has been favorably received and bears out the chemotactic theory is that of Goldscheider and Jakob. About ten years ago these experimenters demonstrated that the injection of bacterial toxins and certain albuminous extracts into the circulation of animals caused a preliminary decrease in the number of the leukocytes and then a secondary hypoleukocytosis. According to these observers the primary hypoleukocytosis is due to a repellant chemotaxis on the leukocytes which causes their temporary arrest in the capillaries. There is then exerted an attractive chemotaxis which causes the stimulation to over-production of the blood-making organs. That there is something peculiar to the nature of the agents or toxins causing the leukocytosis is shown by the fact that in some diseases (*e.g.*, typhoid fever and malaria), the toxins exert an ultimate negative chemotactic influence on the white cells, resulting in hypoleukocytosis.

When we recall that leukocytosis often goes hand in hand with inflammation, and that inflammation is conservative in

* Presented before the Society at the request of Dr. Koplik.

nature, we can readily see that leukocytosis, while often a manifestation of disease, is really an important process which frequently subserves a useful purpose. That leukocytosis is conservative in nature is illustrated by the observations of internists and surgeons that virulent cases of pneumonia and severe cases of appendicitis respectively may frequently be distinguished by the absence of leukocytosis.

A selective as well as a quantitative influence is exerted on the leukocytes by different agents; thus in acute and chronic infections the chief increase is in the polymorphonuclear leukocytes; in trichiniosis and asthma, in the eosinophiles; and in lymphatic leukemia, in the lymphocytes.

As the result of the process of digestion, as the result of hydrotherapy and the administration of certain drugs, and in pregnancy, there occur physiological leukocytoses. While a certain amount of work has been done on digestive leukocytosis, we are not aware that careful and systematic studies have been undertaken on the various physiological leukocytoses that may occur in either adults or children.

An increased number of leukocytes after the ingestion of food is present both in adults and children; it has frequently been observed that from one to two hours after eating the increase begins, reaching the maximum from three to four hours after the meal. Durando Durante has shown that digestive leukocytosis is fairly constant and well marked in young children. This observer found the maximum increase present in the second hour after a meal; the chief increase was in the polymorphonuclear cells. Within certain limits the leukocytosis increased in proportion to the quantity of nitrogenous food ingested. Though in intestinal affections in infants there occurred retardation in the time of the digestive leukocytosis, yet there was no decisive change in its degree. Of interest in connection with this subject is the observation of Luciani, who, in the thirty days' abstinence of the Italian faster Succi, found a marked leukopenia present during the last three weeks of the fast.

During early infancy the normal leukocyte count is different from that in after life. On the normal blood counts in infants and children careful and exhaustive work has been done by Dr. Anna Perlin, whose article on the subject is as elaborate as it is interesting and instructive. The subjects were from one day to sixteen years of age, well nourished, in good general condition, and

with no signs of organic disease; in all cases digestive leukocytosis was avoided and control counts were made. A resumé of some of the excellent work done by this authoress has been arranged in the following table.

Age	Number of Cases	Maximum Leukocyte Count	Minimum Count	Average Count
1 day	13	19,000	13,800	17,100
2 days	13	19,600	16,400	17,800
3 days	16	19,900	12,600	16,300
4 days	5	19,000	14,000	16,960
6 days	4	13,400	12,200	12,800
10 days	3	12,000	10,000	10,970
12 days	2	12,000	9,800	10,900
17 days	1	11,600	11,600	11,600
3d and 4th week	3	13,200	9,900	11,700
10th week to 18th month	12	15,000	8,800	10,900
2d year to 4th year	16	13,400	8,200	10,000
5th year to 7th year	16	11,200	7,800	9,400
8th year to 16th year	11	9,280	7,000	8,500

Dr. Perlin quotes the results of other observers and finds that in the main her findings correspond with those in the literature. Though all authorities are agreed that the leukocyte count is normally very high during the first three days of life, yet her own results are somewhat lower than those of other writers. It will be seen from the above table that from the first week of life to the fourth year, the leukocyte count, though not high, is yet slightly higher than the normal count in adults.

Another feature of prominence in infant life concerning the leukocytes is the fact that physiological variations depending upon certain temporary influences are more marked than in adults. So also with pathologic variations in infants; leukocytosis, as well as temperature, diarrhea and other signs of departure from the normal, can in infants be produced by slight morbific influences, which in adults would fail to produce these signs.

It is well known that in connection with the study of the blood in pneumonia patients, leukocytosis is an important as well as an interesting feature; and especially interesting is the study of leukocytosis in the pulmonary affections of children. In an article on "The Leukocyte Count in the Diagnosis of Diseases of Children," Head gives the results obtained in 15 pneumonia cases by Stengel and White, and himself. In all of the 3 cases of bronchopneumonia and 12 cases of lobar pneumonia there was a pronounced leukocytosis. Head found that the count is lower in bronchopneumonia than in lobar pneumonia, and that in the latter

type of disease the leukocytosis appears to be more pronounced in proportion as the child is younger.

In "A Report of 8 Cases of Pneumonia in Infancy, Treated with Antipneumococcic Serum," J. L. Morse says that the blood examinations in his cases "were too few to be of any value and the results were, moreover, not uniform." Interesting were his findings in an adult case of pneumonia in which the leukocyte counts were more frequently made. A drop in the leukocytosis usually followed the injection of the serum, and on the whole there was a continued diminution in the leukocyte counts while the injections were kept up; omission of an injection would be followed by a slight raise in the count.

Reports of work like that of Head above mentioned are not numerous in the literature, and the few that we were able to find were based upon an insufficient number of cases to be of much value. Yet considerable work in this direction has been done in the hospitals; at Mount Sinai Hospital, for instance, Dr. Koplik has always emphasized the importance of leukocyte counts, and in pulmonary cases especially has always insisted upon careful and frequently repeated blood counts being made by the internes.

The observations in this paper are based upon the records of 50 cases of pulmonary diseases in children, which occurred in the service of Dr. Koplik and myself.

I. LEUKOCYTE COUNTS IN BRONCHOPNEUMONIA.

There were 19 cases ranging in age from ten months to five and one-half years (see Table I. at end of article). The highest leukocyte count taken during the height of the illness was 73,000; the lowest, 12,600; the average was 33,900. There were twelve recoveries and seven deaths. In the former all the counts during the first few days of the illness were high, while toward the end of the illness the counts were low except in 2 cases; of these, 1 was complicated by mastoiditis and the other by tonsillitis. All of the 7 fatal cases gave high counts early in the disease; shortly before death the counts in 4 of these cases were markedly diminished, while they remained high in 3 cases; of these latter 3, 1 had an extension of the process and 1 was complicated by pertussis.

On comparing the age of the patients with the degree of leukocytosis, it will be seen that in children under two years of

age the leukocytosis is comparatively low. Inspection of Table I. shows also that the leukocytosis was independent of the extent of pulmonary involvement. In 5 of the cases only a small portion of one lobe was involved, yet the counts were high; and in the cases of extensive involvement (diffuse bronchopneumonia) the counts were comparatively low. Furthermore, it will be seen from the table that in those cases in which the temperature was higher than 103° F., the blood counts were usually lower than 30,000; while in those cases in which the temperature was lower than 103° F., the counts were usually higher than 30,000. It would appear, therefore, that the degree of leukocytosis and height of temperature bear no direct correspondence.

II. LEUKOCYTE COUNTS IN LOBAR PNEUMONIA.

There were 24 cases studied, varying in age from two and one-half to nine years. The highest count, while the pneumonia was active, was 55,800; the lowest, excluding the post-typhoid case and that of pleurisy, was 20,200; the average count was 31,700. It is interesting to note that of the 2 cases in which there was no leukocytosis, 1 was preceded by typhoid fever and the other was accompanied by pleurisy. There were but 2 fatal cases, and in both a well marked leukocytosis was present during the height of the disease, while just before death a marked drop in the leukocyte count was noted; one of these patients was suffering with aspiration pneumonia following post-diphtheritic paralysis.

Though a well marked leukocytosis is present when only one lobe is consolidated, yet the counts are even higher if more than one lobe be involved. Thus, leaving out of consideration the case following typhoid fever, the 7 cases in which more than one lobe was consolidated all showed a very high leukocytosis (30,600 to 55,800, average 39,800).

In lobar pneumonia as well as in bronchopneumonia, the leukocytosis seems to bear no relationship to the temperature. There may be higher temperatures and comparatively low initial counts, or there may be low temperatures and high initial counts.

During the course of the disease an increasing leukocytosis is the rule, reaching the maximum just before the crisis and then dropping rapidly. The marked fall that takes place is illustrated by the following cases selected from our series:—

	After the onset.	After the crisis.	Drop.
Case 1	26,400	8,000	18,400
Case 3	47,200	17,000	30,200
Case 12	33,000	12,000	21,000
Case 14	50,000	12,000	38 000
Case 22	36,000	9,200	26,800

In lobar pneumonia of adults there is frequently observed a precritical drop in the leukocyte count, upon the value of which as a confirmatory sign of approaching resolution some authors lay considerable stress. In the lobar pneumonia of children our own observations show that this precritical drop is frequently entirely absent. Moreover, a considerable transient fall in the leukocyte count owing to other causes sometimes occurs long before the time of resolution, and on the other hand there occasionally occurs a continuance of the leukocytosis for some time after the crisis. We believe, therefore, that in children the sign referred to is too inconstant and unreliable to be of any great prognostic significance.

On the other hand we have found that the leukocyte count is of great diagnostic aid in cases of lobar pneumonia and pleurisy with effusion in which it is suspected that empyema will supervene. We can recall cases of lobar pneumonia in children in which complete resolution had occurred and the temperature and leukocyte count had dropped to normal, but in which there remained signs of a small amount of fluid at the base of the affected lung. Careful and frequently repeated counts were made in these cases, and a sharp rise in the leukocyte count before other signs and symptoms had appeared was regarded as sufficient indication to warrant an attempt to confirm our suspicions by exploratory puncture of the chest; and, indeed, we were not disappointed. In such cases, therefore, we regard the rise of the leukocyte count as at least strong presumptive evidence in favor of the supervention of empyema.

The claim of various writers that the leukocytosis in pneumonia is modified by the virulency of infection and the power of resistance of the patient and their assumption that these two conditions determine the white cell count and ultimately the prognosis of the disease, seems to us in the present state of our knowledge rather speculative; for as yet there is no precise method of measuring and reducing to a numerical scale the "virulency of infection" and "power of resistance." And in the animal world it has been shown that while ultimate leukocytosis may follow an inocu-

lation of a given strength, yet this leukocytosis does not increase proportionately with the strength of the inoculating material. As yet we have no bacteriological means of gauging with any degree of accuracy the grade or degree of bacterial infection in the human economy, nor have we any positive tests for gauging the resisting power of the patient. True, we have certain clinical data to guide us in determining whether a given case reacts mildly or vigorously; but these clinical phenomena—*e.g.*, heart's action, degree of fever, cerebral and other complications, number of lobes affected, etc.—are too indefinite in nature to be reducible to a mathematical problem that will enable us to estimate or predict a blood count. In the disease under consideration, moreover, we have an infection that may be due to one or more of a variety of bacteria. In one instance it is due to the pneumococcus of Fraenkel, in another to the bacillus of Friedlander, while the streptococcus, the staphylococcus aureus and albus, the bacillus pyocyaneus, the bacillus of influenza, and Eberth's bacillus can all produce pulmonary inflammations of varying types and intensities. The fact that different micro-organisms can cause different kinds of pneumonia seems to us an indication for careful examinations of the sputum in all cases of this disease in which there is expectoration, with a view of aiding in the differentiation of the kind of pneumonia and possibly determining by this means the severity of the particular case. When this aid to diagnosis becomes universally practicable it may then be that we shall be able to speak more intelligently about the virulency of infection.

III. LEUKOCYTE COUNTS IN EMPYEMA.

In all of the 7 cases there was marked leukocytosis at the onset. There were six recoveries and one death; the fatal case gave the highest blood count at the onset of the disease and the highest terminal count. In one of the cases that recovered the process was bilateral; yet the leukocytosis was no higher than in the unilateral cases.

CONCLUSIONS.

(1) A well-marked leukocytosis is present in the bronchopneumonia of children.

(2) Leukocytosis in bronchopneumonia is independent of the amount of lung involved.

(3) The degree of leukocytosis in bronchopneumonia stands in no relation to the height of the temperature.

(4) Though there are some exceptions, yet in bronchopneumonia the general rule is that failure of the leukocyte count to drop when the pulmonary signs disappear indicates either a complication or a fatal termination of the illness.

(5) A constant and considerable leukocytosis may regularly be expected in the lobar pneumonia of children. The degree of leukocytosis is about the same as in bronchopneumonia (bronchopneumonia: average leukocytosis in 19 cases, 33,900; lobar pneumonia: average leukocytosis in 24 cases, 31,700).

(6) The leukocytosis in lobar pneumonia differs from that in bronchopneumonia in that the white blood count is higher when the pulmonary involvement is greater. If in lobar pneumonia two or more lobes be involved, a relatively high blood count may be looked for.

(7) The leukocytosis in lobar pneumonia furnishes no clue as to the height of the temperature.

(8) An increasing leukocytosis is the general rule in the lobar pneumonia of children, reaching the maximum just before the crisis. While failure to drop before the crisis may indicate a complication, yet this may be of no special significance. The precritical drop in the lobar pneumonia of children is inconstant, of little or no prognostic value, and cannot be utilized as means of determining the time of crisis.

(9) There is a high leukocytosis at the onset of empyema in children.

(10) In general it may be said that the diagnostic value of the leukocytosis in the pulmonary affections of children is limited.

(11) In certain instances, however, the leukocyte count is of great diagnostic aid. When, for example, in lobar pneumonia, resolution and the drop in the leukocytosis have occurred, and there are present signs exciting suspicion that empyema will be a sequela, then blood counts should frequently be made at regular intervals. A sharp rise in the count, provided that other causes of leukocytosis can be excluded, is then strong presumptive evidence of a supervening empyema.

For aid in the preparation of the following tables, the writer is indebted to Dr. Leo Kessel, house physician at Mount Sinai Hospital.

TABLE I.

BRONCHOPNEUMONIA.

Case	Age	Weight	Site of Lesion	Rectal Temperature	Pulse	Respirations	Complications	Leukocyte Count	Result
	2 years, 3 months	26 lbs., 4 oz.	R. U. and L. L. lobes	101°–105°	140–160	36–44	Otitis Media	27000–8600	Recovery
2	5 " 7 "	33 " 9 "	R. U. and L. L. lobes	101°–103°	140–110	68-32	Mastoiditis	12600–13000	"
3	4 " 7 "	35 " 4 "	L. L., R. M. and R. U. lobes	99°–104°	110-150	28–44	None	73000–10200	"
4	4 " 6 "	37 "	L. L. lobe	99°–103°	110–140	28–36	Acute Follicular Tonsillitis	63200–20800	"
5	5 "	39 "	R. U. and L. L. lobes	100°–104°	100–130	30–48	None	24200–12200	"
6	3 "	30 " 6 oz.	R. M. and R. U. lobes	99°–105.6°	80–140	28–50	"	16600–4000	"
7	3 " 8 months	33 " 9 "	R. U. and R. L. lobes	99°–104°	90–160	30–45	"	38000–11200	"
8	13 months	16 " 5 "	R. U. and L L. lobes	99°–105°	96–150	34–50	"	23800–12000	"
9	10 "	12 " 1 "	R. U. and L. L. lobes	99°–103.4°	90–160	30–64	"	22200–14000	"
10	4 years	29 " 8 "	R. U. and L. L. lobes	99°–103°	108–144	30–70	"	52800–27200	Died
11	3 " 3 months	24 " 7 "	R. U., R. L. and L. L. lobes	103°–105°	120–160	36–70	Enteritis	20600–11000	"
12	5 " 2 "	31 " 9 "	R. U. and L L. lobes	103°-105.6°	136–150	45-62	Periostitis of Rib	20000–10600	"
13	4 " 7 "	28 " 4 "	R. U. lobe	102.4°–105°	150–154	46–60	Extension of Process	56000–32000	"
14	2 " 1 month	19 " 4 "	R. U. lobe	100°–103°	120–150	50–60	Pertussis	45000–50000	"
15	1 year, 1 "	21 " 2 "	R. U. and R. L. lobes	100°–106°	140–170	44–66	Empyema	36000–11200	"
16	1 " 2 months	21 " 1 "	R. U. and R. L. lobes	99°–106°	144–180	38–70	Enteritis	42600 9700	"
17	1 " 5 "	18 " 8 "	R. L. lobe	99°–101°	132–116	40-28	None	19800–11600	Recovery
18	2 years, 3 "	25 " 10 "	R. U. and R. L. lobes	99°–103.8°	140–114	38–28	"	29400–12600	"
19	19 months	22 "	L. L. lobe	102.8°–99°	140–104	44–28	"	23000–8600	"

TABLE II.

LOBAR PNEUMONIA.

Case	Age	Weight	Site of Lesion	Rectal Temperature	Pulse	Respirations	Complications	Leukocyte Count	Result
1	4 years, 3 months	36 lbs., 2 oz.	R. U. lobe	105.4°–99.8°	136–106	48–26	None	26400–8000	Recovery
2	8 "	53 " 6 "	R. L. lobe	102.6°–99°	120-96	36–26	Pleurisy with Effusion	20200–9000	"
3	6 "	44 " 9 "	R. U. lobe	104.4°–99°	156–90	70–30	None	47200–17000	"
4	6 "	43 " 4 "	R. L. and L. L. lobes	105°–99°	138–120	38–28	This case followed Typhoid Fever	9600–4800	"
5	5 " 6 months	36 " 5 "	R. U. and L. L. lobes	104°–100°	120–100.	36–26	Empyema (operated)	30600–15000	"
6	5 "	36 " 11 "	R. L. lobe	102°–99°	128–104	54–24	Pleurisy. Tubercle Bacilli not found	8000–6800	"

TABLE II.—*Continued.*

LOBAR PNEUMONIA.

Case	Age	Weight	Site of Lesion	Rectal Temperature	Pulse	Respirations	Complications	Leukocyte Count	Result
7	5 years	37 lbs., 6 oz.	R. U. and R. L. lobes	101.6°–100°	124–100	40–34	Aspirat'n Pneum. following Post-diphtheritic Paralysis	41000–34400	Died
8	2 " 4 months	27 " 7 "	R. U. lobe	105.8°–99.2°	170–100	76–24	None	29800–13600	Recovery
9	4 "	33 " 4 "	L. L. lobe	100°–99°	116–96	32–24	"	25400–11600	"
10	6 "	44 "	R. U. lobe	103°–100°	130–104	32–24	Empyema (operated)	31000–16800	"
11	7 "	49 "	L. L. lobe	106°–99°	176–114	46–34	Pleurisy with Effusion	35200–17600	"
12	6 "	35 " 8 oz.	R. U. and R. L. lobes	102°–99°	130–120	42–26	Empyema (operated)	33000–12000	"
13	3 " 4 months	32 " 10 "	L. L. lobe	104.4°–99°	118–90	36–26	None	33800–10600	"
14	4 " 9 "	40 " 10 "	R. U lobe	105°–99°	140–90	52–24	"	50000–12000	"
15	5 " 8 "	44 " 4 "	L. U. and L. L. lobes	105°–99°	160–116	56–32	"	45000–12400	"
16	2 "	22 " 11 "	R. L. and L. L. lobes	104.6°–99°	138–100	60–44	"	39000–13800	"
17	2 " 4 months	25 " 8 "	R. U. and R. M. lobes	105°–103°	160–150	56–42	"	55800–24600	Died
18	6 " 2 "	44 " 2 "	R. M. lobe	103.6°–98.8°	130–110	40–30	"	20600–10000	Recovery
19	5 " 10 "	43 " 9 "	R. U. lobe	104.6°–99°	130–120	38–30	"	21600–11600	"
20	5 "	36 " 4 "	L. L. lobe	102°–99°	112–98	36–24	"	36400–12000	"
21	3 " 8 months	33 " 7 "	R. M. lobe	103°–99°	132–100	36–24	"	26400–8600	"
22	9 "	58 "	R U. lobe	105°–99°	140–96	52–28	"	36000–9200	"
23	5½ "	31 " 10 oz.	R. L. lobe	105.8°–99°	128–76	58–28	"	27000–12400	"
24	9 "	.....	R. U. and R. M. lobes	105°–99°	130–100	40–28	"	34600–15000	"

TABLE III.

EMPYEMA.

Case	Age	Weight	Site of Lesion	Rectal Temperature	Pulse	Respirations	Complications	Leukocyte Count	Result
1	18 months	21 lbs., 9 oz.	Right side	104°–99°	128–108	36–32	None	25000–11200	Recovery
2	13 "	20 " 2 "	Left side	104°–99°	180–110	80–40	"	34200–10000	"
3	4 years	34 " 5 "	Right and left side	103°–99°	130–100	68–38	"	26400–13800	"
4	3 years, 8 months	34 "	Right side	104°–101°	140–126	38–32	"	33900–15800	"
5	2 "	30 " 6 oz.	Left side	104°–99°	120–108	36–26	"	24000–12800	"
6	2 "	23 " 4 "	Right side	104°–99°	140–108	38–26	Measles	22000–18000	"
7	5 "	39 " 7 "	Left side	105°–102°	152–150	44–40	None	35800–20000	Died

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AN UNUSUAL CASE OF TUBERCULOSIS IN AN INFANT.

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Tuberculosis is a common disease in children, but the following case presents such interesting problems in diagnosis and etiology that it seems worthy of record.

The infant, weighing nine pounds at birth, the second child of exceptionally healthy parents, always lived in the best environment. The family history is good in the direct line, all being healthy and long-lived. The only cases of consumption in the family are remote and in collaterals.

The food of the child was breast milk for the first seven months, at the end of which time cow's milk modified with cereal water was given. The cow's milk was of the best from dairies whose milk was used especially for infants, and whose cows were frequently tested for tuberculosis. The infant received no other food. At the age of nine months, just before it was taken ill, it weighed twenty pounds and six ounces. It had always been a healthy, happy, vigorous baby, with rosy cheeks and firm flesh, and it had never been ill. During the two weeks prior to the attack of vomiting and fever, which ushered in the fatal illness, it had gained a pound and three ounces. Its appetite and spirits were particularly good at this time. In other words, there was no prodromal stage of debility or anemia.

The initial symptoms were sudden fever and vomiting lasting a day, but recurring several days later. The infant became fretful, ground its teeth frequently and rolled its head from side to side. Although there were no catarrhal symptoms, and no acute pain, it was thought best to have an examination made of the ears in the hope that their condition might explain the fever, which had now become continuous. Dr. E. A. Crockett found a slight otitis media of one ear and punctured the drum with the escape of a thin serous fluid. Later he punctured the other ear, and, as the first closed up, this was again opened. There was evident relief of the symptoms; the head rolling stopped, and the infant

slept all night, but the fever continued unabated. Several times later in the course of the disease the ears were examined by Dr. Crockett, but nothing abnormal found, so that otitis as a cause of the fever was ruled out.

Another cause for obscure fever was sought in the condition of the kidneys, but several examinations of the urine showed nothing abnormal. The blood was also examined. The Widal test was negative; there were no plasmodia of malaria; the number of leukocytes was within normal limits, and there was no anemia. In this way several other causes of fever were eliminated. Frequent physical examinations by several consultants, as well as by myself, during the whole course of the disease showed nothing abnormal. The lungs and heart were negative; spleen and liver not enlarged; there was no dilatation of the stomach, and, aside from a varying and rather increased amount of gas in the intestines, nothing abnormal could be found in the abdomen.

The fever varied irregularly from 100° to 104° F., and was often higher in the morning than at night. Several times the temperature sank for a few hours to normal; once or twice becoming subnormal. The general course of the fever was lower during the last two weeks of the disease. At the end, however, it mounted rapidly above 106° F.

After the ears had been attended to, the infant appeared to have but little acute pain, although it often moaned a great deal, and almost never smiled during the whole course of the disease. Its mind remained clear to the last few hours, and its strength and will power were unusual. When it was emaciated to the last degree it persisted in desiring to be sat up, and it would then vigorously hold up its head and sway it from side to side. Until the last its pulse was strong.

The infant's digestion as shown by the stools was good during the whole course of the disease. Smooth yellow movements of perfect consistency occurred even when the temperature reached 105° F. Vomiting was at first only occasional; perhaps once a day; and the amount of food retained was sufficient. During the last month vomiting, and especially disinclination to take any food, became prominent features, but at no time was there vomiting of large amounts of mucus or of food. Bile was often vomited. In other words, there appeared to be no evidence of intestinal or of stomach indigestion, or of accumulation from pyloric

stricture, and it was believed that the vomiting was reflex from the fever. Thorough evacuations with calomel, with the idea that there might be obscure ptomaine poisoning, had no effect on the fever.

During the first half of the illness the food consisted of top milk modified with barley water, but when the vomiting and the disinclination for food began, milk was at first omitted. Cereal water, albumin water, whey and broths were all tried ineffectually. It was found that the infant would retain best what it liked most; that it would gag and immediately vomit anything that was distasteful, and that, as a rule, the vomiting was most frequent when the temperature was highest, although there were exceptions to this rule. During the last two weeks the infant was fed by enemata, an ounce and a half of peptonized milk every four hours. These were well retained until the last and seemed to be the chief support of life, as the infant took and retained almost nothing by the mouth. After an illness of over nine weeks the baby, emaciated to a skeleton, died.

To sum up, we have here the case of an exceptionally large and healthy infant of good inheritance and environment, suddenly taken sick with fever and vomiting. The unexplained fever continues for over two months, and ends in death. During all this time nothing is found on physical examination with the sole exception of a serous otitis during the early weeks of the disease. This is relieved by puncture and does not return. The diagnosis of a septic fever due to germ infection seemed the only reasonable one, and, although tuberculosis was considered, infection by some other organism seemed more probable. The autopsy, by Dr. Oscar Richardson, whose account follows, showed extensive tuberculosis throughout the peritoneal cavity.

These questions naturally arise: Why should a healthy infant with good environment and heredity suddenly be stricken with tuberculosis? What was the source of the infection? and Where the point of entrance?

PATHOLOGICAL REPORT.—By O. Richardson, M.D., Assistant Pathologist Massachusetts General Hospital. At the request of Dr. Townsend I made in March, 1905, a postmortem examination of a case of his from the clinical picture of which only a provisional diagnosis had been made.

The case is of interest, in that so grave a disease process al-

though practically localized within the peritoneal cavity should not give more manifestation of its presence, and further, that the disease process itself should be so peculiarly localized and of such unusual character.

At the examination the body presented was that of a male infant, twelve months of age, exceedingly well developed, but emaciated. On making the primary incision it was at once noticeable that the peritoneal cavity had been practically obliterated by a dense layer of grayish white firm tissue which more or less completely surrounded the abdominal viscera, binding them roughly into one mass. The organs of the body were then removed *en bloc* with the preservation of their continuity, and later, but with great difficulty, the abdominal viscera were finally freed from the investing layer of tissue and the character of the disease process made out.

The thoracic cavity and its organs can be dismissed with a few words. The pericardium, heart and great vessels were not remarkable, the pleural cavities free from fluid and the lungs free from adhesions. The lung tissue on section presented no areas of consolidation and showed nothing abnormal, except for the presence in the tissue of each lung of two minute yellowish nodules rounding up just beneath the pleura. They were about 2 mm. in diameter and, on microscopical examination, proved to be minute foci of caseous pneumonia.

The bronchial lymphatic nodes on section showed nothing abnormal.

The abdominal viscera on section presented the following conditions:

Liver.—The organ was surrounded by the layer of tissue previously referred to, which attained here a thickness of 1 cm. in places. The substance of the organ was fairly firm and presented a yellowish, rather homogeneous, section surface. Microscopical examination showed marked fatty metamorphosis of its tissue, but no areas of tuberculosis. In places the investing thickened layer of tissue about the organ encroached slightly into the liver substance.

Spleen.—Other than for the investing layer of tissue which microscopically extended in places a short distance into its substance the organ was not remarkable.

The pancreas, adrenals, kidneys, ureters and bladder, on section, presented nothing abnormal.

Section of the stomach showed a normal-appearing mucosa; but on laying open the intestine the mucosa at the lower end of the ileum presented in two or three places very small shallow losses of its substance with slightly ragged edges. The mucosa was not remarkable otherwise.

The peritoneal surface of the intestine was generally coated with a thick layer of grayish white, firm, in places fibrous-like, tissue which bound the coils of the intestines together, but without occluding them. The omentum was lost in the investing layer of tissue about the organs and the mesentery was thickened and showed in several places small, grayish yellow masses which on section consisted of firm, homogeneous, caseous-like tissue resembling that of tubercular lymphatic glands. In the situation of the superficial ulcers in the mucosa of the ileum, previously mentioned, the small masses of caseous-like tissue were closely applied to the peritoneal coat of the wall of the intestine and on stripping the investing layer of tissue from the peritoneal coat of the intestine many rather large, tubercle-like masses were disclosed dotting the surface here and there.

Microscopical examination of sections of the investing layer of tissue in the regions of the various organs showed it to consist of sheets of fibro-cellular tissue surrounding large areas of homogeneous, eosin-staining material, the marginal portions of which showed many hematoxylin stained fragments of cells, apparently portions of disintegrated nuclei, which in places were margined by epithelioid-like cells. In other places there were areas of eosin-staining tissue where the forms of the dead cells could be faintly distinguished. No giant cells or definite tubercles were found.

A section of the investing layer of tissue stained with carbol fuchsin and decolorized with 3 per cent. hydrochloric acid showed rather numerous typical tubercle bacilli.

Sections of the wall of the intestine and the caseous, lymphatic, gland-like masses in the mesentery showed in places foci of epithelioid-like cells, with smaller and larger areas of caseation and a few giant cells and resembled the ordinary histological picture of tuberculosis.

The case thus resolves itself into an infection with the tubercle bacillus, probably through the intestinal wall in the region of the

superficial ulceration. The character of the lesions produced place the case in that group in which the tuberculous process spreads rapidly throughout the peritoneum forming a thick fibro-cellular layer with caseous masses throughout its substance and closely applying itself about the viscera, but with little, if any, production of tubercular lesions within them.

The question of the source of the tubercle bacilli in these cases of tuberculosis in infants is of interest.

In the case under consideration, the bacilli in all probability gained entrance to the tissues through the mucosa of the intestine in the region of the superficial ulceration, and must have come from ingested food or material in which the bacilli were present.

It is possible that an infant might ingest infected material as food or with food, but it is more probable that the food itself, that is to say, the milk, contained the bacilli.

This raises the question of the possibility of the infection of man by the bovine tubercle bacillus.

De Schweinitz, Dorset and Schroeder have recently recorded in the bulletin of the United States Bureau of Animal Industry a series of experimental inoculations on hogs, cattle and monkeys with bovine and human tubercle bacilli. As a result of their experiments they find that certain tubercle bacilli of human origin possess quite as great pathogenic power for hogs and cattle as tubercle bacilli of bovine origin and that monkeys are highly susceptible to infection with tubercle bacilli of bovine origin when they are fed with food containing the bacilli. This food in some of the experiments on monkeys consisted of milk in which the bacilli were placed.

This experiment on monkeys approaches as near as possible the conditions under which infants may acquire tuberculosis from infected milk.

In regard to the cultural characters of tubercle bacilli, they state that two of their most virulent cultures of the human tubercle bacillus cannot be distinguished in any way from those of the bovine tubercle bacillus.

The results of this remarkable series of experiments, together with those obtained by many other observers, and which are an essential agreement, seem to make it reasonably sure that in many instances tuberculosis in infants arises from the ingestion of milk infected with the bovine tubercle bacillus.

DISCUSSION.

DR. ROTCH.—Was the tuberculin test used in this case?

DR. TOWNSEND.—It was not. It was thought of, but all of the consultants, as well as myself, considered tuberculosis improbable, and the temperature was so constantly elevated that the test could not have been made.

DR. BLACKADER.—I would like to know the condition of the lymph nodes.

DR. TOWNSEND.—The bronchial nodes were not affected, but the mesenteric were.

DR. BUCKINGHAM.—I recall two brothers that I saw within two weeks of each other, both dying of tuberculosis as we believed. No necropsy was made, as the diagnosis seemed perfectly clear. The family history of both parents was good and the surroundings ideal. The milk used was furnished from a farm kept in excellent condition by the brother of the children's father. The animals had been tested and so we simply gave up the search for the place from which the tuberculosis came. I learned later that a man who worked on the farm was discovered to be in a state of advanced tuberculosis and that he had been freely expectorating about the stable floors. I do not wish to discuss intercommunicability, but we must consider in any given case the possibility of infection of the milk from unusual human sources.

DR. MORSE.—This case, which I saw several times with Dr. Townsend, was most interesting. Each time we talked over the possibility of tuberculosis and excluded it, although it seems now as if we ought to have recognized it. It seems to me we are not at all justified in ruling out tuberculosis in any case because we cannot get a history of exposure. A positive history of exposure is of importance, but a negative history is of little value. An adhesive tuberculosis limited to the peritoneum in an infant of this age is certainly most unusual.

DR. EDSALL.—It seems to me that one cannot properly say that there was no source of exposure to tuberculosis until one has definitely shown that there was no possibility of acquiring tuberculosis through the milk; and, as a matter of fact, it is in most instances almost impossible to determine that the milk was definitely non-tuberculous. I am convinced that the testimony concerning the intercommunicability of human and bovine tuberculosis has grown very positive. A very considerable proportion of the abdominal cases in infants and young children that have been properly investigated in this regard—I think 15 or 20 per cent. of these cases at least—have been shown with the greatest

probability to have been due to bovine infection. Dr. Townsend's case presented lesions that were most suggestive in this connection, and it is in just this type of cases that a bacillus with many of the usual characteristics of the bovine bacillus has been found in a considerable proportion of instances. In a case that I recently saw with Dr. Fife, which he and Dr. Ravenel have since reported, there were extensive lesions of the mesenteric and retroperitoneal glands, and the bacillus obtained from these lesions produced violent and rapid tuberculosis in a calf. I think that this and other observations of Dr. Ravenel, and of other investigators in this country and abroad, show very clearly the probability that young children, whose diet consists largely of milk, suffer infection from milk with sufficient frequency to make it a matter of real importance.

DR. ROTCH.—It is not surprising that the diagnosis could not be made in this case, since we meet with many cases where there is continued temperature and which are not necessarily tuberculosis. In addition to this the temperature so often precludes the tuberculin test that the diagnosis becomes still more difficult. In connection with the general discussion of the subject, I cite the case of a boy who came under my care with tuberculous peritonitis. Laparotomy proved the diagnosis to be correct and he recovered. A feature to be noticed in this case was that the boy had been fed almost entirely upon the milk of a cow which on being killed was found to be markedly tuberculous.

DR. SAUNDERS.—As to the communicability of bovine tuberculosis, it seems to me we shall have to leave Koch alone and proclaim as loudly as we can, the whole profession, that bovine tuberculosis is very communicable to man. The only question that remains to be settled is as to its varying manifestations. This case which Dr. Townsend reports shows a hyperplastic form of limited degree and should remind us of the fact that bovine tuberculosis is a hyperplastic type of the disease.

Another remark I would like to make concerns the etiology. A friend of mine many years ago said that whenever he had a case of tubercular meningitis without obvious connection he looked out for later developments and that within a short time some other member of that family would prove to be tuberculous. I am able to corroborate that observation from my own experience. I remember one case where I had examined the child's father very carefully and found nothing positive, but within a year he died of rapidly progressive tuberculosis.

I would also like to mention a case of tuberculous peritonitis that I saw recently where there was a peritoneal effusion and a dispute about its character. The tuberculin test was employed and the temperature ran up. About a week later the effusion had

diminished about one-half and whether the injection caused that change or not I do not know.

DR. KERLEY.—The case of Dr. Townsend's, and the one reported by Dr. Buckingham, remind me of one that Dr. Holt saw with me two years ago, when a healthy child of eight months developed tubercular meningitis and died within a few weeks of the onset of the first symptoms. It was a matter of interest to us to know the source of the infection, which remained for the time unexplained. One year later the mother came down with pulmonary tuberculosis, and is now in a very low condition. That was a case in which cow's milk was supposed to be the source of the infection; and yet the child evidently got the disease from its own mother.

DR. BLACKADER.—In Dr. Townsend's case the possibility of the infecting bacilli having been derived from human sources has not definitely been excluded, and in my opinion we are not yet warranted in saying of bovine tuberculosis that it is very infectious for man.

DR. HOLT.—At the Babies' Hospital within the last few years we have made a point of having the families from which cases of tuberculosis occurred investigated, and it has been the rule that some other case of tuberculosis has been discovered at the child's home. Sometimes the immediate family have been found perfectly healthy, but an investigation has disclosed the fact that the young child was frequently "borrowed" by the family in an adjoining apartment for the entertainment of some patient there suffering with tuberculosis. Such accidental infection we found very common. Personally I have yet to see a single case where the evidence of the transmission of bovine tuberculosis to the human was convincing; but that there are such cases I do not doubt.

DR. KERLEY.—Dr. Holt's observation is borne out by our dispensary work. I remember one instance, several years ago, of 4 cases coming from one house, and each of the cases had a good family history. On thoroughly investigating the conditions at that house we learned that the janitor of the building had pulmonary tuberculosis in an advanced state. He had taken this position because he could not follow his occupation—that of a carpenter. He expectorated promiscuously about the hallways, and succeeded in infecting four of the child-inmates.

DR. TOWNSEND.—I am very glad that this report has called out so much discussion, and I want it understood that I am myself very doubtful as to the source of infection and do not by any means feel sure that it was due to infection through the milk. The family was naturally anxious to discover the source of in-

fection and we employed every means of research. It is perfectly possible that the child may have taken the germ in by swallowing dust, as we have a great deal of dust in Boston.

Dr. Kerley's case struck me in a different way from what it did him. He spoke of the child dying of tuberculous meningitis and a year later its mother showed tuberculosis. Now the mother was apparently free from tuberculosis at the time the child was taken sick, and she may have been the victim and not the source of infection. The better explanation, it seems to me, is that both of them probably had constitutions that made them especially susceptible to the tubercle bacillus which is so universally common, whose inroads most constitutions successfully resist.

The interesting point to me in this case was that the child had been so healthy and its surroundings so good, and that if healthy children are liable to get such profound infections from tuberculous milk, it seems to me, as Drs. Blackader and Holt have said, strange that we do not see more cases.

A PLEA FOR THE MORE TIMELY USE OF INTUBATION IN LARYNGEAL STENOSIS, AND FOR THE USE OF TRACHEOTOMY IN CERTAIN NEGLECTED CASES.

BY E. W. SAUNDERS, M.D.,
St. Louis.

Since the introduction of antitoxin in the treatment of diphtheria there has been manifested on the part of physicians a strong disposition to postpone interference for the relief of stenosis of the respiratory tract occurring in the course of the disease. In one case related to me recently serum injection was the sole remedy relied upon for the cure of an urgent dyspnea, which was later found to be due to the lodgment of a piece of bone in the larynx.

In my own experience of recent years I can testify that I am seldom called upon to perform intubation until the danger of death by suffocation becomes imminent.

Before the antitoxin era the physician in attendance upon a case of croup would summon skilled assistance as soon as the diagnosis of laryngeal diphtheria was made or suspected.

Now he injects the serum and waits; if the dyspnea becomes more urgent he injects more serum. Finally when he or the family concludes that the patient cannot live many minutes more without relief assistance is summoned.

Undoubtedly the mortality from the disease is kept unnecessarily high by this pernicious custom. Even when the child does not die by suffocation, tardy relief fails to protect it against the dangers of heart failure from overstrain, and of broncho pneumonia, due to the prolonged retention of muco pus in the tubes.

Even if there were no danger in this unjustifiable delay the common dictates of humanity should forbid a medical man from allowing his patient to suffer the most horrible of all forms of torture, slow suffocation.

Those who thus procrastinate in the treatment of true croup not only err in putting a false estimate upon the function of anti-

toxin, but also in failing to take into account all the elements of danger in these cases.

In a given case four factors of immediate danger may be at work, beside the remote dangers:

1. Pseudomembrane, dangerous while attached and, like all foreign bodies, dangerous when detached, either partially or wholly.

2. Swelling of the tissues, always provoked to a greater or less extent by the presence of inflammation of any kind or degree.

3. Laryngospasm, which occurs at uncertain intervals during the course of true croup, as well as that of false croup.

4. Retained secretions.

A neglected case of diphtheritic croup may die from increase of the pseudomembrane, or from its detachment and lodgment in the bronchi, from rapid edema of the tissues, from laryngospasm at a time when recovery seems assured, from suffocation due to the retention of an enormous amount of muco pus below the stenosis, from starvation due to the impossibility of administering food or water, or from heart failure.

My second contention, for the revival of tracheotomy in the treatment of certain cases, will, I fear, receive little favor at the hands of this Society, but I feel constrained to make it because of the lessons learned in actual practice since the introduction of intubation by the lamented Dr. Joseph O'Dwyer.

No one hailed the invention of this American genius with greater delight than I did, or honors his memory more highly to this day, and in making a plea for the substitution of tracheotomy in a small percentage of the cases met with in practice, I am not detracting from the lustre of his fame.

The first class of cases demanding tracheotomy are those of unrelenting spasm of the glottis. I have met with two such, in one of which life was saved by the cutting operation. The only author who speaks of this experience, so far as I know, is Hugues, of Paris, who had several cases in a series of nearly 200 intubations.

It may be true that a more dexterous operator could succeed where others have failed, but still the fact remains that provision should be made for the safety of a small class of cases that cannot be successfully intubated by men of long experience. The second class are those of tracheal or tracheobronchial diphtheria.

If the introduction of the O'Dwyer tube in a child whose reflexes are not yet abolished produces no paroxysm of coughing, and if the relief obtained after waiting some time is slight or imperceptible, then I believe the wisest course is to perform tracheotomy, as would be done in the case of any foreign body lodged below the glottis. It is true that often the eventually loosened tracheal membrane is coughed up against the tube and the two together are expelled, but I believe it is safer to open the windpipe at once and extract all the membrane which is found accessible.

The third class consists of those cases with extensive membranes in the fauces. The occlusion of the tube from above is so instantaneous and inevitably fatal, unless a competent person be at hand to pull out the tube by the thread left attached, that we cannot afford to leave the patient for a moment menaced by such a danger.

Perhaps the forcible removal of all the pseudomembrane in sight would be preferable to tracheotomy, although I have never tried it.

The fourth class of cases in which I would recommend tracheotomy consists of those that are apparently moribund. When a child is found almost pulseless, and making feeble efforts to breathe at lengthening intervals, the introduction of the finger into the fauces will perhaps extinguish the spark of life, and even if the tube be placed instantly, artificial respiration gives very unsatisfactory results in my experience. A quickly executed tracheotomy does not interfere with the respiratory efforts, gives access to the tracheal pseudomembrane, if present, and enables the operator to perform efficient artificial respiration.

In doing the operation quickly and sometimes without skilled assistance, I have found the following method best. The child is held upright facing the light. This does away with all necessity for sponging. After the first incision the sharp hook is inserted into the wind-pipe, which greatly facilitates the further rapid dissection.

When the trachea is opened, and before the sharp hook is withdrawn, a bent hairpin is introduced with the convexity toward the larynx. This is self-retaining, and gives access of air to the opening, whilst it does not interfere with the extraction of membrane below the wound.

In conclusion, I would say that, whatever may be thought of the plea for a limited use of tracheotomy, I hope that this Society will exhort the profession to invoke intubation more often and more promptly than has been done since the dawn of the antitoxin era.

DISCUSSION.

DR. CHAPIN.—I think the plea for earlier intubation is very timely, and I hope it will go before the profession that this Society endorses it. The chances are so much better when it is done fairly early. The time for intubation has arrived when there is sucking in of the soft parts on inspiration. We all know that delay until the child is almost moribund, or the heart very weak, lessens the chances of recovery.

I cannot quite agree with Dr. Saunders as to the very frequent necessity for tracheotomy. If intubation is done earlier the difficulties now occasionally met with will occur much less frequently. I have watched many cases at the Willard Parker Hospital, and tracheotomy is rarely done; practically only in those cases where the child keeps coughing up the tube. There are cases where it is almost impossible to keep the tube in, even those tubes specially devised for the purpose.

The condition of an excess of membrane above the tube is one I have not seen. I am very glad to have attention called to it, and shall watch for it, but I should think it largely a theoretical rather than a practical suggestion.

In most cases the child will cough the membrane out through the tube. There is a tube made especially for cases in which the membrane is pushed down before the tube—a short round tube with a large calibre—and it seems to me any membrane that will get through the average opening in the trachea will pass through that tube. And I must confess that I should hesitate to do a tracheotomy in the way the doctor has described, with the child in an upright position.

Will you tell us how you get in; is it below the isthmus?

DR. SAUNDERS.—No; usually above.

DR. CHAPIN.—I am not a surgeon and have not operated for a good while, but I should think it difficult to do it that way.

DR. BOOKER.—I wish to add my testimony to the plea for early intubation in laryngeal diphtheria. There is no doubt of a growing tendency to put off intubation, and in many cases until it is too late to do any good. It is better to reserve the child's strength and save suffering by introducing the tube as soon as difficult breathing is apparent. I cannot agree, however, with Dr.

Saunders in regard to the substitution of tracheotomy for intubation in moribund cases. I have had no difficulty in inserting the tube in such cases, and I am sure that I could not have succeeded with tracheotomy. In a case in which the tube was removed on the fifth day, difficulty in breathing began soon after I had left the house. When I saw the child six hours later, she was lying on the bed in a room by herself, where the parents had left her under the impression that she was dead. At first sight I thought this impression was correct. The child was comatose, her face purple, and body perfectly limp, with gasping respiration at long intervals. The tube was inserted as she lay upon the bed, without assistance, and in a few minutes later she was in good condition and breathing easily. The introduction of the tube in this case reminded me of my first practice in intubation on the cadaver.

Similar cases have, no doubt, come to most physicians who have had much experience in intubation. I do not know of anything that gives more happiness to the physician than to witness the sudden relief from most intense suffering, and restoration from the dying state, which often happens from intubation in moribund cases of laryngeal diphtheria.

There is a class of cases which have not been referred to by Dr. Saunders in which tracheotomy may be preferred to intubation. In some of the cases where the tube has caused injury to the larynx and cannot be removed without danger of suffocation, it may be of advantage to substitute tracheotomy. I have never had but one case of this kind, and this case I believe was due to failure in following O'Dwyer's advice to use the smaller tube. The uncertainty of getting to the child in a reasonable time in case the tube should be coughed up, caused me to use a large tube which required some force to pass into the larynx. Tracheotomy was resorted to, and when I last heard from the child some months later, she was still wearing the tracheal tube.

DR. MCCOLLOM.—I was very much interested in Dr. Saunders' paper and heartily agree with him regarding early intubation. There is a tendency to wait too long. When there is difficult breathing and rigidity of the sternocleidomastoid muscles, it is useless to wait longer. If the child does not sleep well and has this muscle stiffness I always put in a tube. Regarding the class of cases that come to tracheotomy, there is one class in which the intubation tube does not do any good, that is where there is a good deal of swelling about the tonsils and pharynx, but these cases are comparatively rare; I should say that out of 1,500 intubations, I have seen only 5 or 6 cases of this sort. I cannot agree with the doctor about tracheotomy in the moribund cases. Tracheotomy may be simple or difficult. I have seen many children brought into the hospital gasping for breath, where I have

intubed in much less time than I could have done tracheotomy. The children recovered, whereas they would have died had I tried to do tracheotomy.

A retained tube is usually due to traumatism at the time of its introduction. When a child coughs up a tube frequently, it is probably better to do tracheotomy. I recall one case where the child wore an intubation tube for five or six weeks and yet tracheotomy had finally to be done.

I do think, and I want to emphasize the fact, that we ought to do intubation early. There is a good deal of literature on the inhalation of steam, slaked lime, etc., but they do not do any good. There is one thing to be said about intubation, that perhaps it is not as good for private as for hospital practice, because in an institution there is somebody at hand to perform tracheotomy if the tube comes out or becomes obstructed. I remember a case I had some years ago where, after I had introduced the tube, it remained about a minute and was then coughed up with a membranous cast of the right and left bronchus.

If the intubation tube does not give very marked relief within twenty minutes, the only thing to do is to take it out. Frequently the child will then cough up a considerable amount of membrane and it may not be necessary to put the tube back.

DR. ADAMS.—I do not think we can emphasize this point of prompt intubation too strongly. The unwillingness on the part of a physician to take the warning to which attention has been called was the cause of a death I witnessed last winter. The harm that can result from intubation is so slight that the operation should be done the moment obstruction is noticed. Intubation really helps the antitoxin by permitting the entrance of plenty of fresh air and allowing nature to cast off the membrane that has formed.

As regards the moribund cases, I saw a case last winter, and while waiting for the surgeon to arrive, I thought the child was dead, and, in fact, the father was confident of its death, but Dr. Richardson, having arrived, introduced the tube and in a few moments we had the gratification of seeing the child breathing.

We should go forth and emphasize the importance of this position, that the moment we see the suprasternal depression, or the moment the sternocleidomastoids stand out prominently, intubation should be done without regard to the use of antitoxin.

DR. MCCOLLOM.—I want to emphasize that point regarding the rigidity of the sternocleidomastoid muscles, because you can feel that, and often see it, before there is any suprasternal depression.

DR. CHAPIN.—Do you regard the sinking of the larynx as of any importance?

DR. MCCOLLOM.—No, sir; it is a later symptom.

DR. CRANDALL.—One of the most satisfactory cases in my practice is one of those apparently moribund children whom I intubated without even the use of the gag. We ought to give all of these cases the benefit of the doubt.

DR. CHAPIN.—I would like to say a word about artificial respiration in this connection. In 1 or 2 cases that almost died we brought about good results by simply standing the child on its head.

DR. BOOKER.—I have had good results in these cases from pouring a small quantity of whisky and water into the throat, some of which passed into the trachea and brought on coughing, which was soon followed by good breathing.

DR. ABT.—Are there not some specimens of congenitally narrow larynx that will not permit the entrance of a tube because no tube is obtainable that is sufficiently small?

DR. KOPLIK.—I shall show such a pathological specimen tomorrow.

A CASE OF ACUTE YELLOW ATROPHY OF THE LIVER IN A CHILD FIVE YEARS OF AGE.

BY A. H. WENTWORTH, M.D.,

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The following brief description of acute yellow atrophy of the liver is taken from Quincke's article in Nothnagel's Spec. Pathologie und Therapie, Bd. XVIII.:

Acute yellow atrophy of the liver is a rare disease. It occurs more frequently in females than in males, and is most likely to occur in young adults between twenty and thirty years of age. Children are very rarely affected. Merkel[1] reported 18 cases and Schmidt[2] reported 16 cases occurring in childhood from birth up to ten years of age.

Cause.—Nothing definite is known of the cause. It is generally accepted that the alterations in the cells are due to a protoplasmic poison. These changes are most marked in the liver, but other organs are also affected. The source of this hypothetical poison has not been determined. Similar appearances in the liver and other organs can be produced by the action of a number of substances. Acute atrophy of the liver has been known to occur after various acute infectious diseases, and in syphilis. It has followed acute alcohol poisoning, the ingestion of mushrooms and sausages, and prolonged chloroform narcosis. Phosphorus poisoning produces the condition if the patient survives for several weeks. It may occur in livers which are normal or in livers which are affected by chronic processes, such as cirrhosis or passive congestion.

Symptoms.—As a rule, the symptoms of the first stage of the disease are those of catarrhal jaundice of a mild type. The earliest symptoms are usually those of gastric catarrh, loss of appetite, coated tongue and vomiting. After a few days jaundice occurs. At times jaundice is delayed. This stage lasts for a few

1. Merkel, *Münchener Med. Woch.*, 1894, s. 89.
2. Schmidt, Dissertation, Kiel, 1897.

days or for several weeks. The second stage of the disease develops suddenly in most cases, and is shown by the occurrence of mental symptoms. There is usually somnolence alternating with restlessness; delirium and even maniacal symptoms; vomiting; possibly convulsions; increasing coma, and death within a few days. Recovery is rare when these symptoms occur. During the second stage there is a rapid diminution in the size of the liver; tenderness in the liver region; enlargement of the spleen. There may be vomiting of blood; nose bleed; intestinal hemorrhage; hemorrhages from the genito-urinary tract and ecchymoses in the skin. The urine contains bile pigment; generally a small amount of albumin and, as a rule, leucin, tyrosin and other abnormal metabolic products. Urea is much diminished. Ammonia is usually increased. Sugar is not present. The temperature remains normal or subnormal. Towards the end of life the temperature may become elevated.

The diminished liver dullness which occurs in the second stage is due in part to the reduced size of the organ, and in part to the flabby consistency of the liver which permits it to collapse and to drop back under the diaphragm.

Hemorrhages occur in a majority of cases in the second stage of the disease. The commonest seats of hemorrhage are in the stomach and the skin. As a rule, the quantity of blood vomited is not large, and either resembles "coffee grounds," or is brownish red and does not coagulate. Convulsions occur in about one-third of the adult cases and in almost all cases in children. There may be clonic spasms of groups of muscles, or tremors, or general clonic spasm. The pupils are generally widely dilated in this stage and react imperfectly. It is possible for the mental condition to improve temporarily.

Duration.—The disease may last for from a few days to eight months. Thierfelder's statistics showed that 50 per cent. of the fatal cases died between five and fourteen days, and 30 per cent. between three and five weeks. The second stage is short, as a rule it lasts for from one and a half to three days. It very rarely lasts longer than one week. In protracted cases the nervous symptoms are not so severe and a long second stage usually occurs only in favorable cases.

Diagnosis.—A diagnosis in the first stage is usually impossible. The occurrence of unusual or severe general symptoms in

the course of an otherwise mild attack of jaundice might lead one to suspect the disease. In the second stage, the occurrence of severe nervous symptoms; a rapid diminution in the size of the liver; enlargement of the spleen, and the presence of leucin and tyrosin in the urine, would in most cases suffice for a diagnosis. The sequence of symptoms as shown by the history, the absence of fever, the intensity of the jaundice, the diminished size of the liver and the presence of leucin and tyrosin in the urine, would serve to distinguish cases of acute yellow atrophy from cases of socalled "icterus gravis"; a condition that has been found associated with various bacterial infections.

In cases of phosphorus poisoning there may be a history of the ingestion of phosphorus followed by acute gastric symptoms. Then a pause of two days, followed by jaundice on the third day. The jaundice is less intense and the liver is enlarged and very tender after the third day. The cerebral symptoms are more often those of depression corresponding to the prostration. The hemorrhages are more profuse and widely distributed. The duration is less than one week.

Prognosis.—In most cases the disease is fatal. The prognosis is worse when the cerebral symptoms are severe and occur early. Cases occurring in pregnancy are always followed by miscarriage and are almost invariably fatal. At one time the possibility of recovery was doubted. Recent investigations, however, have shown that recovery does occur at times. More or less regeneration of liver tissue occurs in all cases of acute yellow atrophy and it is probable that the destruction of liver tissue is not so excessive in cases that recover. Bauer[3] reported a case of acute yellow atrophy that recovered, and three months later the patient died of miliary tuberculosis. In this case the diagnosis of acute yellow atrophy was substantiated by finding at the autopsy marked evidences of regeneration of the liver tissue.

Treatment.—Even if the disease were suspected in the first stage, it is very doubtful if any treatment would avail. Quincke suggests the usual treatment in cases of catarrhal jaundice, with the addition of remedies designed to produce intestinal antisepsis. In the second stage, the treatment is symptomatic.

The case which I am about to describe was seen by me in consultation with Dr. E. E. Allen, of Charlestown. The diag-

3. Verhandlung des Congresses f. Innere Medicin, 1893, s. 185.

nosis was not made during life. The patient was a boy five years of age. The family history was negative; both parents and two other children living and well. At eighteen months the patient had tubercular peritonitis from which he recovered without operation in about six months. During the following year and a half he gained steadily in weight and at the end of that time appeared normal in all respects. At the age of three and one-half years he had pneumonia of the left lower lobe. The disease followed the usual course and terminated in nine days without complications. In February of the present year he and one of his sisters had an attack of catarrhal jaundice. The boy recovered in about a week. The sister's attack lasted somewhat longer. From that time until the present illness he appeared to be perfectly well. He weighed forty-eight pounds and was very active and vigorous. The present illness began on April 19th. The first symptom noticed was slight jaundice. This was followed in a day or two by vomiting and a slight degree of malaise. The appetite remained fairly good throughout his illness. The temperature was never found to be elevated. The bowels were constipated. The child was about the house and out of doors until within four days of his death. The symptoms were those of a mild attack of catarrhal jaundice. On the night of May 5th, the sixteenth day of his illness, he appeared to be delirious. At intervals during the night he cried out and tried to get out of bed. When restrained he was quite violent. I saw him on May 6th. At this time his condition was as follows:

He was well developed and large for his age. He was somnolent, but roused when examined. The skin was quite deeply jaundiced. The mother stated that the jaundice had increased very much within twenty-four hours. The pupils were equal, moderately contracted and reacted to light. The patella reflexes were not obtained. The pulse was full, slightly irregular, 96-100 in a minute. The respiration was normal. The rectal temperature was 99.4°F. The muscles were relaxed. There was no rigidity nor tenderness in the neck. No paralysis. The throat was reddened. A lymph node at angle of jaw on right side 2 cm. in length. Otherwise lymph nodes not enlarged. The right drum membrane was slightly thickened and opaque. The heart and lungs were negative. The abdomen was soft, not distended, not tender; tympanitic, except over descending colon. Over this

portion of the intestine percussion was dull and fecal masses could be felt. Otherwise nothing abnormal was detected in the abdomen. The liver dullness began at the sixth rib in the mammillary line and the edge of the liver was felt 1½ cm. below the border of the ribs in the same line. The upper border of splenic dullness was not clearly defined. The spleen was not palpable. Genitals, bones and points negative. There was no eruption nor edema in the skin. A small quantity of urine was obtained and examined by Dr. Allen for albumin and sugar, with negative results. From the child's behavior during my examination I concluded that he probably had not been delirious during the preceding night, as the family believed, but that the symptoms were due to irritability. He had had no movement from the bowels for three days and he was intensely jaundiced, and I believed that this combination could account for his somnolence in the absence of symptoms pointing toward any other condition. A fat-free diet was prescribed, together with several doses of phosphate of sodium, in conjunction with enemata. Sodium bromide was suggested if the symptoms demanded it.

The subsequent history is as follows: The bowels were induced to move freely and the child appeared to show slight improvement. He was quieter during the night of May 6th than on the previous night. Early in the morning of May 7th, however, he became delirious and insisted upon turning out the gas. He was violent when restrained. Once he was allowed to get up, to see what he would do. He started at once for another room, but stumbled against the furniture and would have fallen had he not been assisted. He was given a dose of codeia which quieted him. After 9 A.M., on May 7th, the stupor gradually changed to unconsciousness. At one o'clock there was a profuse hemorrhage through the nostrils of dark-colored blood, which showed no tendency to clot. The mother then remembered that on the previous afternoon on two occasions the child spat up blood-tinged fluid. Dr. Allen was called and remained with the child until his death, three hours later. Dr. Allen stated that the child was completely unconscious and of a pallid yellow color. The pulse was not much accelerated at any time, but remained slightly more than 100. It was strong and could be counted at the wrist up to the time of the patient's death. The respiration was rather superficial and gradually became irregular. There

was cyanosis toward the end and marked tonic contractions of the muscles of the body and of the extremities. It is probable from the history, the character of the blood and the condition of the pulse that death was not due to hemorrhage, but that the blood had collected gradually in the stomach.

A post-mortem examination was made twenty-four hours after death. The undertaker had injected formalin into the femoral artery and into the abdominal cavity a few hours after the child's death.

The head was not opened.

Thorax.—The parietal and visceral layers of the pleura are adherent by fibrous tissue over the lower two-thirds of the left lung. The lung tissue is not noticeably abnormal.

The pericardium is not abnormal. The heart is of normal size and consistency. The cavities are empty. The valves and endocardium are not abnormal. Nothing abnormal is detected in the aorta or in the esophagus. The bronchial lymph-nodes are of normal size and appearance.

Abdomen.—The omentum is adherent in several places by fibrous tissue to the anterior parietal wall. In these adhesions are two caseous masses about 2 cm. in diameter. On section these caseous masses are found to be partly calcareous. There are fibrous adhesions between the liver and omentum. The liver and spleen are so firmly adherent to the diaphragm by dense fibrous tissue that they cannot be detached. The peritoneal layer of the intestines is free from adhesions and appears to be normal. The weight of the liver, which includes the portion of diaphragm which is adherent, is 660 grams. The liver is *not diminished in size.* The embalming of the body has affected the liver so as to render it firmer and somewhat paler than it otherwise would have been. On the inferior surface of the liver, there is a dense caseous lymph node, and a small caseous mass between the liver and the diaphragm. The surface of the liver is smooth, except where the diaphragm is adherent. The cut surface of the liver presents areas of a bright greenish yellow color on a background of dark red. The size of these greenish yellow areas varies. Close inspection shows them to consist of foci, round or oval in shape, measuring $1\frac{1}{2} \times 1\frac{1}{2}$ mm. to 1×2 mm., each showing in its centre a depression (central vein?) and in its immediate periphery a zone of darker color. These foci are sepa-

rated by a narrow line of reddish grey color. Sometimes the foci occur singly or in pairs, more often a number are grouped together to form larger areas, ⅓ to 1 cm. in diameter, of a general greenish yellow color. In the central part of the right lobe near its superior surface, a large mass (3 x 5 cm.) is so formed, though here there are small intervening areas of reddish color. In other places are similar, though smaller, areas. Except these greenish yellow areas, the liver tissue appears homogeneous, and of a dark red color. In one vein there is an occluding dark red clot, not attached to vessel wall.

The gall bladder is of normal size and contains tenacious pale yellow mucus. The common duct is narrowed about half way between the gall bladder and the duodenum. At this point it admits a small probe about 2 mm. in diameter. On section at the narrow portion there are two or three transverse elevations of mucous membrane. A lymph node 1½ cm. in diameter is adherent to the outer portion of the duct at the narrow portion.

The *spleen* is much enlarged. The weight of the spleen, together with a small piece of adherent diaphragm, is 140 grams. The consistency is firm and the surface is smooth. The cut surface is thickly beset with pale grayish rounded foci, averaging 1½ mm. in diameter, on a background of dark rich red. These foci are very uniform in size and shape and evenly distributed over surface of organ. None show any softening of centres.

The stomach is of normal size and the lesser curvature is adherent to the under surface of the liver by fibrous tissue. This portion of the stomach is dark red in color and rather friable. The mucous membrane of the stomach in the region of the lesser curvature is dark red and friable. On the posterior portion of this area, near the cardiac orifice, are three ragged tears extending down to the muscular layer. They average ½ cm. in length, without loss of substance, so that the edges can be approximated. They are evidently caused by breaking up the adhesions during removal of the stomach. The stomach contains dark fluid blood.

Each kidney weighs 100 grams: The capsule strips easily without tearing kidney substance. The cut surface of the kidney is pale and slightly bile-stained. The cortex averages 5 mm. in thickness; the glomeruli are visible as small grayish points; the pyramids are darker than the cortex and the markings are fairly distinct.

The intestines are contracted from the action of the formalin. About four feet from the stomach and for about four feet of the length, the lumen is filled with tarry blood. Otherwise the intestines are normal.

Some of the mesenteric lymph nodes are enlarged and two or three are caseous and calcareous. The pancreas is not abnormal.

There are no hemorrhages other than those already described.

The organs were taken to the Harvard Medical School for microscopic examination. The following report was obtained from Dr. W. T. Councilman:

"Sections from the red areas show no liver cells. The topography of the lobules is preserved and around the periphery of each lobule there are numerous newly-formed bile ducts. The tissue is composed chiefly of loose fibrillæ of connective tissue enclosing connective tissue cells and groups of plasma cells. The capillaries can be fairly well distinguished. The extraneous cells lie between them, taking the place of the liver cells. The yellowish green areas are composed of more or less altered liver tissue. No necrotic liver cells can be distinguished. The interlobular tissue is increased and infiltrated with lymphoid cells. The capillary bile ducts are dilated and filled with yellowish green bile casts.

"The most striking feature of this case is the extreme degree of destruction of liver tissue with the absence of macroscopic signs of atrophy.

"Sections of the kidney show great swelling of the cells which are filled with fat vacuoles. Spleen shows intense congestion and hemorrhage in the pulp, and hyperplasia of the follicles. The enlarged follicles are composed almost entirely of lymphoid cells, scattered among which are numbers of large phagocytic cells. There is, however, no marked phagocytosis."

It will be seen that the onset, symptoms and course of the disease in this case were perfectly typical. On the other hand, the evidence obtained by physical examination was quite misleading. The liver was rather increased than diminished in size and was palpable during the last stage of the disease. In spite of its normal or *slightly increased* size, the liver cells were destroyed in at least three-quarters of the liver. Thus the size and consistency of the liver were quite unlike what is usually decribed as characteristic of acute yellow atrophy. The spleen,

which was very much larger than normal, was adherent to the diaphragm in such a position that it could not be palpated. The disease was not suspected and so the urine was not examined for leucin and tyrosin.

It is interesting to note that the tubercular process that had been present in infancy appeared to be completely arrested.

DISCUSSION.

DR. GRIFFITH.—I had the fortune to see, a few years ago, a case of acute yellow atrophy in a boy of seven years. He had been taken ill about two weeks before with slight jaundice, which gradually became intense. The liver was said to have been enlarged at first, but later to have grown smaller. About two days before he was admitted to the hospital he became comatose. We made the diagnosis of acute yellow atrophy, having been fortunate in obtaining a specimen of urine, in which leucin and tyrosin were found in abundance. Postmortem examination showed a very great diminution in the size of the liver. However, on microscopic examination, it did not show as great a degree of atrophic change as we had expected to find.

THE RESULTS OF DECAPSULATION OF THE KIDNEYS FOR NEPHRITIS IN CHILDREN, WITH REPORT OF A CASE IN A CHILD OF TWENTY-SIX MONTHS.

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In a careful study of American and foreign literature for decapsulation of the kidneys for nephritis, the writer has been impressed with the fact that very few cases in children have been reported, the large majority of operations having been performed upon adults. In this paper, the results of decapsulation for nephritis only, will be considered. Pain in the kidney, movable kidney, and surgical affections of the kidney will not be dealt with.

It seems already proven that such cases are suitable for operation and this paper considers only cases of nephritis uncomplicated by such conditions. The following cases have been found in the literature:—

CASE I.—F. G. Balch (*Boston Medical and Surgical Journal*, 1904, p. 90). Mary B., twelve years old; edema of feet, legs and eyes; some peritoneal dropsy; previous history of measles, mumps and scarlet fever as a child; scanty secretion of urine varying from 10 ounces to 2 ounces in twenty-four hours; albumin, ½ to 1 per cent.; urea, 3.59 per cent.; sediment typical of parenchymatous nephritis; death in five days after operation. Case would probably have died if not operated on.

CASE II.—Francis H. J., thirteen years old; scarlet fever at four years with edema of face and legs during convalescence, symptoms persisting for two weeks; never strong after scarlet fever; was known to have nephritis for perhaps nine months before operation. Had a systolic murmur; edema of extremities; feet cold and clammy; at time of operation, albumin, ½ per cent.; fine and coarse granular casts with fat and renal cells. Operation on April 12th, both kidneys decapsulated. On May 28th, albumin, $^1/_{10}$ per cent.; a rare hyaline and finely granular cast. October 15th, no albumin; specific gravity, 1.013; no casts;

had slight edema of face and hands two months ago, lasting twenty-four hours. Child evidently much improved since operation.

CASE III.—J. C. Hubbard (*Boston Medical and Surgical Journal*, 1904, p. 94). Boy nine and one-half years old; has had measles, diphtheria and typhoid, not scarlet fever; edema present; urine smoky; acid, 1.010; albumin, ⅛ to ¼ per cent.; many hyaline and granular casts; blood; renal cells; few fatty and epithelial casts. Tapped three times, with death nineteen days after operation.

CASE IV.—Augustus Caillé (ARCHIVES OF PEDIATRICS, 1902, p. 734). Child five years old; chronic parenchymatous nephritis for three years, following an attack of measles. Child not cured by medical treatment. Operated on February 15, 1902, with decapsulation of both kidneys. February 4, 1903, no casts; no red or white cells in urine; albumin, 0.05 per cent. April 16, 1904, specific gravity, 1.021; no albumin; no sugar; no casts. Child pale but in good health.

CASE V.—J. W. Elliott, in the *Boston Medical and Surgical Journal*, 1902, p. 457, reports case of a boy thirteen years old. In the first report, no improvement was noticed, the case being reported shortly after operation. Marked improvement two months after operation. Doing very well seven months after operation. Two years after operation, in good health; no casts, and only a trace of albumin in the urine.

CASE VI.—Dr. E. Willys Andrews (*Annals of Surgery*, 1904, p. 617). Child nine years old with severe, well advanced chronic parenchymatous nephritis. Patient had been watched for some years; symptoms had slowly but progressively grown worse; anasarca extreme; albumin, ½ to 2½ per cent; operation in September. At first there was slight improvement; albumin diminished to 1 per cent. Casts remained numerous; anasarca never improved. Child remained in hospital three or four weeks, then went home, after which the albumin returned to the same amount as before operation. Dr. Andrews considers the case not improved by operation.

CASE VII.—Dr. C. H. Frazier (*University of Pennsylvania Medical Bulletin*, 1903, 1904). Child, aged nine years, had scarlet fever at four years. Nephritis with albuminuria and general anasarca at the age of five. Symptoms disappeared rapidly under

treatment; has had two relapses with more or less complete recovery. Chronic parenchymatous nephritis steadily growing worse under best medical care; marked anasarca and ascites; average daily quantity of urine 22 ounces, loaded with albumin and casts. In one week, after first operation on right kidney only, anasarca entirely disappeared, and quantity of urine greatly increased. Two months later, the left kidney was operated on. Child is apparently in good health. It lived in a much improved condition for over a year after the operation, when she took cold, acquired another attack of acute nephritis and died in the course of a couple of weeks. Autopsy was refused.

CASE VIII.—Dr. A. Primrose, in the *Montreal Medical Journal,* 1904, p. 317, reports the case of a boy ten years old, with chronic nephritis; albumin, 1.6 per cent.; numerous hyaline, granular and epithelial casts; paracentesis abdominis performed seven times in the six months preceding the operation. His general health remained good for one year and nine months after decapsulation, when the symptoms of nephritis returned. He improved under treatment. Ten weeks later another acute exacerbation of his nephritis occurred, and a second operation for decapsulation was performed with apparently no benefit. Two months later, another relapse, and one month later, still another relapse. No report as to death or recovery.

CASE IX.—Dr. Primrose also reports a boy eight years old; had measles in infancy; diphtheria seven months before operation. Known to have nephritis two months. No improvement under medical treatment for one month. Specific gravity, 1.040; albumin, 1.5 per cent.; hyaline, fatty, granular and epithelial casts. Right kidney decapsulated. Death fifteen days later from uremia.

CASE X.—Rotch and Cushing (ARCHIVES OF PEDIATRICS, August, 1903) report a boy nine and one-half years, with advanced parenchymatous nephritis; albumin, $\frac{1}{8}$ to $\frac{1}{3}$ per cent.; urea, 5 to 9 grams in twenty-four hours; hyaline, granular and fatty casts. Patient steadily growing worse. Symptoms temporarily relieved by decapsulation. Death eighteen days after operation.

The history of my own case, Case XI., is as follows:—Pearl Glickman, aged twenty-six months; family history good; child was born at full term, raised on the breast and had good health until seventeen months of age, when she had a mild attack of scarlet fever. Three weeks later dropsy developed in face, hands and

feet. This disappeared largely, after three weeks of treatment. Two weeks later, dropsy returned and, in spite of systematic and careful medical treatment, persisted. The urine at this period contained a large amount of albumin and many casts. Her condition grew gradually worse, and she was admitted to the Jefferson Hospital October 8, 1904.

On admission the child was pale and the extremities cold and bluish. She exhibited general anasarca, the face being so much swollen that the tips of the eyelashes barely protruded. It was entirely impossible for her to open the lids, and there was peritoneal dropsy. Heart's action rapid; sounds are less distinct than normal; no cardiac murmur; lungs normal. Blood examination made October 9th, is as follows:—

Erythrocytes	3,180,000
Leukocytes	14,000
Hemoglobin	64 per cent.
Color index	1 " "
Polynuclear	89 " "
Small lymphocytes	10 " "
Large lymphocytes	1 " "
Numerous poikilocytes	

She remained under my medical care for a week, and during this time a systematic use of hot baths, diaphoretics, dieretics, laxatives and a liquid diet failed to be of benefit; in fact, the child grew progressively worse, and during the forty-eight hours preceding operation showed distinct uremic symptoms. Death seemed imminent unless a radical change for the better should occur quickly. Dr. W. W. Keen was asked to see the child with the hope that a decapsulation of the kidneys might offer some chance of saving its life. The parents were informed that the child might die under the operation, and their consent being obtained Dr. Keen operated, with the assistance of Dr. Joseph Hearn, on October 15th, Dr. Keen operating upon the right kidney, while Dr. Hearn operated on the left. The edema of the back was so great as to obscure almost entirely the spines of the vertebra. Dr. Keen removed a small portion of the right kidney by transverse incision, the microscopical report of which appears later in this paper. Both kidneys were a dark purplish color and distinctly larger than the normal. Both capsules were easily

separated and pushed thoroughly forward anteriorly and posteriorly.

The child stood the operation remarkably well, chloroform being the anesthetic used. Twenty-four hours after the operation the child showed a most marked improvement, the dropsy was distinctly less in the face, arms and legs. The eyes could easily be opened, and the child was hungry. It was difficult to estimate the total quantity of urine passed in the twenty-four hours, the major portion of it being lost in the child's napkins.

On October 17th, the patient was distinctly better; dropsy almost completely disappeared, and the child sleeps well. Three days after the operation the amount of urine voided was very distinctly increased. On the fourth, fifth and sixth days after decapsulation, the dropsy returned to a moderate extent, the child became drowsy and evidently was not doing so well. Improvement then began, and, with the exception of the period when she was absent from the hospital, January 5 to 18, 1905, there has been no dropsy. Twelve days after the operation she had a slight attack of pulmonary congestion, which rapidly disappeared under treatment.

On October 31st, a small, whitish patch was noticed on the conjunctiva. The child was sent to the out-patient ophthalmological department, and it was found, upon examination, that there were present whitish patches of degeneration in both foveal regions, similar to those of chronic albuminuric retinitis. She was discharged from the hospital January 5, 1905, in very good condition. On January 18th she was readmitted to the hospital with slight edema. She did not receive proper care at home. Under appropriate medical treatment and diet the child rapidly improved, has been in the hospital ever since and, to all external appearances, seems perfectly well.

Dr. Howard Hansell reports, on June 10, 1905, a thorough examination through the medicinally dilated pupil with the electric ophthalmoscope, during ether anesthesia, showing the circulation of the retina to be unchanged from the normal; no hemorrhages or edema to be found, and no pathological changes in the foveal regions.

The child seems perfectly well, is free from all dropsy, is not anemic and is bright and cheerful.

In the study of the subject of decapsulation of the kidneys, it

seems advisable first to consider the method by which this operation is supposed to cure. Edebohls claims that a cure is largely brought about by the increased amount of arterial blood which the kidney receives after the operation, and claims to have found postmortem blood vessels of considerable size entering the kidney tissue, the development of such blood vessels being subsequent and dependent upon the decapsulation. Ziegler says: "When a portion of renal epithelia has been destroyed by a morbid process which spares the interstitial structures, the loss is in general, soon made good by regenerative proliferation of the remainder, and if the circulation is adequately maintained, the new epithelia presently become capable of carrying on secretory function."

Edebohls says: "Renal decapsulation is performed with the object in view of creating new and liberal supplies of arterial blood to the diseased kidney. The denuded kidney and its fatty capsule are most liberally supplied with blood vessels. Both are brought together by the operation over the entire surface of the kidney, and new and large blood vessels form between the kidney and the surrounding fatty tissue. The normal capsule of the kidney forms a barrier, of course, to this new supply of blood made possible by a decapsulation."

If this increase of blood supply to the kidneys, which Edebohls claims to occur, actually does occur, then the increased amount of blood carried to the kidney should improve the circulation in the kidney, and assist in removing waste products. The portion of the kidney not diseased would take on an increased function, and the disease might be checked. It is a debatable question, however, whether the newly formed blood vessels going to the cortex from the fatty capsule are permanent. They are probably only temporary, and it is doubtful, therefore, how much increased blood supply is really carried to the kidney after decapsulation.

A factor which probably has considerable to do with the improvement which is noticed during the first few days after a decapsulation is the massage which the kidneys receive at the hands of the surgeon during the operation. It seems plausible to believe that this necessary manipulation temporarily relieves the congestion and inflammation which exists.

There is much, however, in the study of cases operated upon to lead one to believe that the factor which has most to do with the remarkable improvement that is sometimes noticed after de-

capsulation, is the relief of the kidney tension which is brought about by the removal of the capsule. Experimental decapsulation done on animals by W. H. Gifford (*Boston Medical and Surgical Journal,* July 14, 1904) seems to show that "there is a certain amount of intracapsular tension in undecapsulated kidneys, normal or with nephritis. There is an immediate increase in size of decapsulated kidneys, persisting up to one month at least, afterwards; a decrease to approximately normal size complete at the end of six months. The increase in size is due primarily to the increase in blood supply, possibly resulting from the removal of the capsule."

It is now, I think, universally admitted that the decapsulated kidney becomes enveloped in a much thicker capsule than existed previous to a decapsulation, in from three to four months after the operation. Where the kidneys have been really seriously damaged by nephritis, an improvement may be noticed for possibly a few months after the operation, and it seems easy to understand how the improvement could continue after the formation of the new capsule, which, according to the studies of Dr. Johnson, of San Francisco, and others, seems positively to occur in a few months.

The favorable progress which is at times noted during the first few months following the operation takes place, in the opinion of the writer, before the new capsule is formed, and the improvement which has occurred during these first few months may, in certain cases, be sufficient to produce changes of a character that may be more or less permanent. This gain may continue progressively for perhaps some months after the capsule has been reproduced, and possibly be permanent. Wolff believes that the parenchyma of the kidney is never regenerated. The undiseased cells may hypertrophy and take on partially the function of the destroyed cells, but he does not believe that the old cells are replaced by new ones. However, the probabilities of new cell development seem certainly to be infinitely greater in children than in adults.

Evidence seems to be steadily accumulating against the acceptance of Edebohls' theory of increased blood supply in the kidney being the beneficial factor following the operation, and considerable testimony has accumulated pointing to the improvement being due to the relief of renal tension. Whether the latter is or

is not true does not alter the fact that certain cases do improve in a most remarkable manner after the operation, and it is the duty of the physician to study these cases and select those suitable for operation. If it is possible to separate earlier the cases likely to result fatally from those where a favorable prognosis is probable, either etiologically, or by the history of the case, or by the urinary findings, an earlier operation might in these cases show a decided decrease in the total mortality.

Chronic interstitial nephritis associated with arterial sclerosis, perhaps with changes in the heart and liver, does not seem to the writer to offer much prospect of a cure from a decapsulation; the kidney condition in these cases is usually only a part of a general degenerative condition of the entire body. It is possible that a certain amount of temporary improvement in the kidney might follow an operation in these cases, but a permanent cure, or even a lengthening of life, does not seem to be at the most more than possible.

The removal of the kidney capsule in these cases may assist in temporarily checking the sclerosis of the kidney, but the formation of the new capsule in a few months, and the impossibility of a complete cure effected during these months of new capsule formation, seem to argue against a permanent cure in this class of cases. It is, however, not always an easy matter to decide how much of the inflammation is parenchymatous, how much is diffuse, and how much interstitial.

A careful study of the cases operated upon seems to show that if the nephritis is of comparatively recent origin, the urinary evidences of the nephritis being, perhaps, only of some months' duration, and if under the best medical care the case is not improving but growing progressively worse and there is more or less renal insufficiency, in these acute or subacute cases, a rapid improvement may follow a decapsulation. The symptoms in some of the reported cases disappear as if by magic, and the urinary findings show such a marked improvement that there is no doubt that the operation, and the operation alone, is accountable for such improvement; and there is no reason why, in cases of this character, an occasional complete cure might not result. Marked uremic symptoms add greatly to the danger in these cases, and should cause one to give a very guarded prognosis.

A true nephritis is probably rarely, if ever, one-sided. In 500

autopsy records reported by Guiteras, in all nephritis was found to exist in both kidneys. It seems to be the growing practice, in view of this fact, to operate on both kidneys, although it is the general belief that an operation performed on one of the kidneys assists in the function performed by the other.

Just what part renal mobility plays in the symptoms of the child, it is difficult to say; probably it is a factor of very little importance. If this is true, it makes the successful cases in children all the more noteworthy, as the movable kidney associated with nephritis, albumin and casts in the urine, constitute a distinct class of cases in adults where the operation is followed by good results. In considering the question of mortality, Guiteras, after a careful investigation of 120 cases, gives the following results:—16 per cent. cured; 40 per cent. improved; 11 per cent. unimproved; 33 per cent. deaths. The mortality in chronic interstitial nephritis was 26 per cent.; the mortality in chronic parenchymatous nephritis 25 per cent., and the mortality in chronic diffuse nephritis 75 per cent. Dr. A. R. Elliott, of Chicago (*Medicine,* 1904, page 251), gives the mortality in 76 cases as 47 per cent., including chronic parenchymatous nephritis, and chronic interstitial, the cases showing improvement as 34 per cent.; unimproved 16 per cent.; worse after operation 2.6 per cent, or a total of 65 per cent. not favorably influenced. He is emphatic in his condemnation of the operation.

Both the mortalities of Guiteras and Elliott consist largely, if not entirely, of adult cases. When we come to consider the mortality in children, I find that the results are distinctly better, as is shown later in this paper.

It is important, when possible, to make a careful bacteriologic examination of the urine collected by catheterization of the ureters, for the bacteria present may decide the question of a decapsulation or a nephrotomy.

In considering this question of mortality, it is very important to remember that the large majority of cases reported are hospital cases, and it is just this class of cases where it is extremely difficult to continue careful, systematic and appropriate treatment, many of these children leaving the hospital to return to their homes, where proper treatment is often altogether impossible, and the possible cure, therefore, rendered extremely doubtful. This is well illustrated in my case, where the child did very well in the

hospital, was allowed to go home, became rapidly worse and again quickly improved after its return to the hospital.

The mortality of Bright's disease, where there is disease of the fundus of the eye, is exceedingly high. Dr. G. F. Suker (*New York Medical Journal,* June 14, 1904) says: "It matters not what form of Bright's disease you may consider, the fundus lesions which are of paramount clinical significance are albuminuric retinitis, and neuroretinitis, with or without hemorrhages. This albuminuric retinitis is of the gravest prognostic importance, and that in an inverse proportion to the age of the patient. When present, irrespective of the stage or type of the Bright's disease (excluding the puerperal and scarlatinal types), the operation of decapsulation is absolutely contraindicated. Retinal changes frequently accompany chronic interstitial nephritis, next in frequency the parenchymatous and finally the diffuse variety of Bright's disease. Very seldom in a purely acute nephritis, can a retinitis albuminurica be positively established (not including the puerperal or scarlatinal).

"Patients with albuminuric retinitis rarely live over two years after the retinitis is recognized. Decapsulation only hastens their death, and has not saved a single life in this class of cases that I am aware of. Suker reports 20 cases of decapsulation with retinitis with twenty deaths."

It is a well-known fact that albuminuric retinitis is quite rare in children, and when present, is not nearly so grave a symptom as when present in the adult. Moreover, albuminuric retinitis is seldom seen in the acute forms of nephritis. The number of cases operated upon in children is too small to as yet be able to collect any data in regard to this point, and I have been unable to find any record of the exact percentage of cases of nephritis which show any evidences of albuminuric retinitis. It seems, however, to be rather in the child's favor that retinitis occurs less often than in the adult, and when it does occur, it is not so unfavorable. It probably points to the fact that the kidney lesions in the child are less likely to be associated with other lesions in the body, and hence decapsulation on a child may offer better chance of cure than an operation done in a similar case in an adult.

In my series of 11 cases, there were five deaths, or 45.4 per cent. Of these five deaths, Case VII., operated upon by Dr. C. H.

Frazier, lived in a much improved condition for a year and died from an attack of acute nephritis. One case was distinctly improved by operation. There were four probable, or at least possible, complete cures, or 36.3 per cent., namely, Cases II., IV., V., and XI. Two cases were not improved. All of the cases would probably have died if not operated upon.

The form of nephritis most benefited by operation is the acute and subacute cases, but only those should be operated upon which are not doing well under appropriate medical treatment. The results in the chronic cases is not favorable. Regeneration of the kidney tissue in the child is more likely to occur, and the kidney lesions in the child are less likely to be complicated by other degeneration in other portions of the body, and hence, improvement possible in the first few months following decapsulation is more apt to be permanent in the child.

The eye ground changes are less significant in the child than in the adult.

It is important to collect statistics from private practice, as the results ought to be better than from cases in hospital practice.

URINARY REPORT OF PEARL GLICKMAN, AGED TWENTY-SIX MONTHS.

Date.	10-8-04.	10-14-04.	10-25-04.	11-16-04.	12-10-04.
Color	Amber	Amber	Amber	Light amber	Pale yellow
Specific gravity	.....	.....	.....	1.007	1.010
Reaction	Acid	Acid	Alkaline	Alkaline	Acid
Albumin	¼ Moist layer	Trace	Present	Decidedly present	0.2% present
Sugar	Not found	Not found	Not found	Not found	Not found
Urea	1.3%	2.5%	0.5%	1.2%	1.8%
MICROSCOPICAL.					
Crystals	Triple phosphates	Not found	Not found	Triple phosphates	Not found
Amorphous	Urates	Urates	Urates	Phosphates	.....
Epithelial cells.	Few squamous	Squamous and Columnar	Squamous and Columnar	Spuamous and Columnar	Few
Leukocytes	Numerous	Numerous	Numerous	Few leukocytes	Few
Erythrocytes	Not found	Not found	Not found	.....	.....
Casts	Granular	Granular	Granular	Granular	Epithelial Non-granular
	Hyaline	Hyaline	Hyaline	Hyaline	Finely granular Hyaline

Date	1-20-05.	2-4-05.	3-8-05.	3-25-05.	6-16-05
Color	Straw	Amber	Amber	Light amber	Light yellow
Specific gravity	1.006	1.007	1.014	Not enough urine	1.018
Reaction	Acid	Acid	Acid	Acid	Acid
Albumin	Decided trace	Decided trace	Faint trace	Not found	Decided trace
Sugar	Not found	Not found	.....	Not found	Not found
Urea	.7%	.9%	1.6%	2 8%	1.2 grains per fluid ounce
MICROSCOPICAL.					
Crystals	Not found	.....	.....	Not found	Not found
Amorphous	Urates	.....	.....	Few urates	Trace urates
Epithelial cells.	Many squamous	.....	.....	Not found	Not found
Leukocytes	Many	.....	.....	Few	Very few
Erythrocytes	.....	.....	.....	.....	.....
Casts	Granular	Granular	None	.....	Very few finely granular
	Hyaline	Hyaline	None	.....	Very few

Blood—few red cells. Tube casts not found.

BLOOD REPORT—JUNE 16, 1905.

Erythrocytes 5,220,000
Leukocytes 9,600
Hemoglobin (Dare's hemoglobinomiter). 58 per cent.
Color index

Differential:—

Polymorphonuclear leukocytes ..... 74 per cent.
Small hyalin leukocytes ..... 12 " "
Large hyalin leukocytes ..... 4 " "
Eosinophiles ..... 10 " "
Mast cells. Two were found not typical

October 10, 1904.

Patient.—Pearl Glickman, Philadelphia.
Surgeon.—Prof. W. W. Keen, Jefferson Hospital, City.
Specimen.—Tissue from right kidney.

Specimen consists of a small piece of tissue 2.5 cm. in length; weight, 0.6 gm. The tissue mass resembles in shape the quarter

of a sphere and presents three eliptical surfaces of about equal size and shape, the greatest breadth of any one being 0.8 cm. Two of these surfaces are flat, irregular cut planes, showing no special features, and are pinkish in color. The third is smooth, regular and convex, covered with a thin layer of whitish homogeneous tissue and possesses no macroscopic pathologic lesions. The tissue is tough and firm but not hard in consistency.

The specimen was fixed, infiltrated, blocked, cut, mounted and stained according to approved laboratory methods.

Microscopically, sections show a small triangular area possessing the histologic structure of the kidney cortex. The striæ of the medullary rays or the pyramids of Ferrein can be very clearly distinguished from the labyrinth with its numerous malpighian bodies and tortuous uriniferous tubules of varied calibres. One side of this triangular area is convex and bordered by the fragmentary remnants of a distinct layer of fibrous tissue, which evidently was the capsule of the kidney, but has been, to a large extent, stripped away. The other two sides are straight and cut without regard to any structure. The malpighian tufts are the seat of several distinct, but often associated, changes; in some the tuft is abnormally cellular, the new elements possessing the characters of recently proliferated cells. Other glomeruli are rather shrunken and the glomerular space occupied by a finely granular, faintly acidophilic substance, evidently the solid portion of an exudate which before fixation was liquid. Here and there whole glomeruli have dropped out, leaving their capsular nests vacant. The flat cells lining the capsule of Bowman, are at points, desquamated and in other tufts, granular. The epithelial lining of the uriniferous tubules is swollen, cloudy, or granular, and in some places in active desquamation. The cells are, for the most part, cuboidal or low columnar, and where approaching the normal, possess large distinct nuclei; in some areas the cell outlines are distorted and the nuclei indistinct or unrecognizable. At some points, the connective tissue between the tubules is slightly swollen, and in some fields a distinct, but not abundant, intertubular lymphoid cell infiltration is present. Minute blood vessels are fairly abundant in the section, and here and there small areas of rhexis are present; the latter are more noticeable near the periphery on the convex or capsular side of the section.

Diagnosis.—Acute diffuse nephritis.

DISCUSSION.

DR. HUBER.—Another reason for improvement in some of these cases lies in the fact that quite an amount of serous fluid drains away from the incision. It is on a par with what occurs when incisions are made for the relief of edema. Dr. Peabody has related to me an extremely interesting case where a number of decapsulations were made at varying intervals and he tells me that the capsule was on one occasion restored in six to eight weeks.

I want to speak also of a boy of about ten who was under my care for extensive edema and ascites, with effusion in the pleural cavity whose nephritis followed scarlet fever several years previous. In tapping we found opalescent fluid with milky-fat globules. All sorts of medical remedies were tried without improvement. He had been repeatedly punctured and the limbs enveloped in gauze and much serous fluid drained away. He was tapped several times, and finally, as our remedies were of no avail, Dr. Brewer was requested to do a double decapsulation. He improved wonderfully after the operation, the chylous ascites disappeared without further tapping, and I saw him one year later with no return of the edema, although he still had the evidences of chronic diffuse nephritis.

DR. PISEK (guest).—I would like to add something to the case reported by Dr. Caillé. I saw that case several months ago, now several years since the operation, and the patient is perfectly well.

DR. GRAHAM.—I have only to add that I was very much struck, while studying this subject, to find that in the literature there were reports of only 11 cases of children operated on, while hundreds of adults have been treated. Everything points to the fact that better results might be anticipated in children than in adults, and I want to put in a plea for the operation upon children that are going from bad to worse, that they should be given the benefit of the doubt.

A CASE OF UNCINARIASIS IN A CHILD.

BY SAMUEL S. ADAMS, M.D.,
Washington, D. C.

In presenting this subject to the Society I must admit my ignorance of the disease owing to the rare opportunities for observing its clinical characteristics. To my former pupil, at one time the Resident Physician in our Children's Hospital, Dr. Bailey K. Ashford, U. S. A., great praise must be given for the knowledge we now possess of uncinariasis, especially in Porto Rico.*

The master workman in this field, however, is my colleague in the Georgetown University, Prof. Ch. Wardell Stiles, Ph.D., who, in his report upon the "Prevalence and Geographic Distribution of Hookworm Disease (uncinariasis or ankylostomiasis) in the United States," † has opened a wide field for observation upon the causation of hitherto unexplainable anemias in childhood. I quote his "Summary" in its entirety, believing it to be the best and most concise description of uncinariasis.

"Convinced from theoretical deductions that hookworm disease (uncinariasis) must be more or less common in the South, a trip from Washington, D. C., to Ocala, Fla., stopping at penitentiaries, mines, farms, asylums, schools, and factories, the fact was established that the chief anemia of the southern rural sand districts is due to uncinariasis, while clay districts and cities are not favorable to the development of this disease.

"In the old world, hookworm disease was probably known to the Egyptians nearly 3,500 years ago, but its cause was not understood until about the middle of the nineteenth century, when it was shown to be due to an intestinal parasite, *ankylostoma duodenale.* Until 1893 no authentic cases of this disease were recognized as such in the United States, but between 1893 and 1902 about 35 cases were diagnosed. In 1902 it was shown that a distinct hookworm, *uncinaria Americana,* in-

* Report of the Commission for the study and treatment of "Anemia" in Porto Rico. December 1, 1904, Bureau of Printing and Supplies, San Juan, P. R.

† Public Health and Marine Hospital Service of the United States Hygiene Laboratory, Bulletin No. 10, Washington; Government Printing Office, 1903.

fests man in this country, and this indicated very strongly that the disease must be present, although not generally recognized. It is now established that, in addition to the few cases of old world hookworm disease imported into the United States, we have in the South an endemic uncinariasis due to a distinct cause, *uncinaria Americana.* This disease has been known for years in the South and can be traced in medical writings as far back as 1808, but its nature was not understood. Some cases have been confused with malaria, others have been attributed to dirt-eating.

"The hookworms are about half an inch long. They live in the small intestine, where they suck blood, produce minute hemorrhages, and in all probability also produce a substance which acts as a poison. They lay eggs which cannot develop to maturity in the intestine. These ova escape with the feces and hatch in about twenty-four hours; the young worm sheds its skin twice and then is ready to infect man. Infection takes place through the mouth, either by the hands soiled with larvæ or by infected food. Infection through the drinking water may possibly occur. Finally, the larvæ may enter the body through the skin and eventually reach the small intestine.

"Patients may be divided into light cases, in which the symptoms are very obscure; medium cases, in which the anemia is more or less marked, and severe cases, represented by the dwarfed, edematous, anemic dirt-eater. Infection occurs chiefly in rural sand districts. Above the frost line the symptoms are more severe in summer than in winter, and whites appear to be more severely affected than negroes. Persons who come in contact with damp earth are more commonly infected than others, so that the disease is found chiefly among farmers, miners, and brickmakers. Severe cases are more common in women and children than in men over twenty-five years of age. Uncinariasis is a disease which occurs in groups of cases, and if one case is found in a family the chances are that other members of the same family are infected.

"The testimony of patients severely infected is unreliable. Recalling that any one or more symptoms may be absent or subject to variation, it may be noted that the period of incubation (at least before the malady can be diagnosed by finding the eggs) is from four to ten weeks. Stages are not necessarily distinctly defined, but are described as (1) stage of purely local symptoms,

corresponding to the light cases; (2) stage of simple anemia, corresponding to the medium cases; and (3) dropsical stage, corresponding more or less to the severe cases. The duration of the disease after isolation from the source of infection has been traced for six years and seven months; how much longer infection will last is not established. If a patient is subject to cumulative infection, the disease may last five, ten, or even fifteen years, and in case of light infection perhaps longer.

"*External Appearance.*—In extreme cases there is a general lack of development; skin waxy white to yellow or tan; hair is found on the head, but is more or less absent from the body; breasts are undeveloped; nails white; external genitalia more or less rudimentary; face anxious, may be bloated; conjunctivæ pale; eyes more or less dry, pupil dilates readily; membranes pale, according to the anemia; teeth often irregular; tongue frequently marked with purple or brown spots; cervical pulsations prominent; thorax emaciated; heart beats often visible; abdomen frequently with 'pot belly'; extremities emaciated, frequently edematous, and with wounds or ulcers of long standing.

"*Urine* 1.010 to 1.015; in advanced cases albumin without casts; acid or alkaline. *Feces* reddish brown, contain eggs, and may contain blood.

"*Circulatory System.*—Anemia pronounced, according to degree and duration of infection; blood watery, with decreased red blood corpuscles and with eosinophilia; 'heart disease' very commonly complained of; hemic murmurs present; pulse 80 to 132 per minute.

"*Temperature.*—Subnormal, normal, or 101° or 102° F.

"*Respiratory System.*—Breathing may be difficult, slow or increased to as high as 30.

"*Muscular System.*—Emaciation and great physical weakness.

"*Digestive System.*—Appetite poor to ravenous; abnormal appetite often developed for pickles, lemons, salt, coffee, sand, clay, etc.; pain in epigastrium; constipation or diarrhea.

"*Nervous System.*—Headache, dizziness, nervousness, mental lassitude, and stupidity.

"*Genital System.*—Menstruation irregular or absent; if present, it occurs chiefly in winter; there is a marked tendency to abortion.

"DIAGNOSIS.—The safest plan is to make a miscroscopic examination of the feces to find the eggs; or if feces are placed on white blotting paper, a blood-like stain will be noticed.

"TREATMENT.—Thymol, or male fern (often with calomel); iron, and good food. *Prevention.*—Treat all cases found and dispose of feces.

"PROGNOSIS.—Good, if patient is not too far gone at time of treatment. *Lethality.*—Not yet determined.

"Economically, uncinariasis is very important. It keeps children from school, decreases capacity for both physical and mental labor and is one of the most important factors in determining the present condition of the poorer whites of the sand and pine districts of the South.

"The disease is carried from the farms to the cotton mills by the mill hands, but does not spread much in the mills; nevertheless, it causes considerable amount of anemia among the operatives."

CASE.—James McK., white, aged twelve years, a native of Maryland, but at present residing at 953 Twenty-fifth Street in this city, was admitted to the Children's Hospital, D. C., May 3, 1905.

The family history shows that his father died with a "congestive chill" (?); his mother and four brothers are now in good health; and tuberculosis among his mother's relatives.

The boy's history shows that he was a breast-fed, healthy child and that he has had none of the diseases of childhood. About eighteen months ago he was brought to this city from St. Mary's County, Md. He admits that before coming here he played a greater part of the time in the dirt, but denies eating it. During his residence here he has spent much of his time in the street, but has neither worked nor attended school. About Christmas, 1904, he began to complain of illness. The first symptom of the present illness was that of feeling tired, would sleep much of the day and would not play as formerly. Soon he complained of almost continuous headache and great muscular weakness. His skin soon assumed a peculiar yellow tint, which became more marked from day to day. These symptoms gradually increased and when he was admitted to the hospital (May 3d) he was so weak that he could not walk. During the past few months his appetite has been poor and his bowels have usually

moved every third or fourth day, but he has not had either nausea, vomiting or pain in the abdomen. Nothing abnormal is noticed in his mental or nervous system; nor has he had any fever during his illness. He sleeps well. There is no history of cold or cough. He has had neither chills nor "night sweats," but has lost weight during the past few months.

Physical examination on admission. Height and weight less than normal. Facial expression anxious but intelligent. Nervous system apparently normal, although the boy is rather quiet and sleeps more than is natural.

The muscles are well developed, save a slight wasting. The osseous system is normal.

The skin has a well-marked yellowish tinge over the entire body, but there is neither eruption nor hemorrhage into it. The sclera is clear; the conjunctivæ pale, almost white. The lips are pale and waxy. Neither liver nor spleen is enlarged. The lymph glands are not enlarged. The nails are colorless and the fingers pale. The teeth are sound, the tongue pale and slightly coated, and the breath foul. The stomach is not enlarged. There is neither distention of nor pain in the abdomen. The apex-beat of the heart is in the fifth intercostal space and the dullness is slightly increased toward the left nipple line. The first sound is heard and synchronous with it is a soft blowing murmur, best heard at base, but not transmitted beyond cardiac area. The second sound is distinct. The murmur is undoubtedly hemic.

The pulse varies from 100 to 120 and is soft and compressible. Temperature 99.5° F., per rectum. The lungs are apparently normal, the respirations number from 24 to 32, and there is no shortness of breath.

The genito-urinary system is normal; and the urine is passed normally.

Urinalysis:—Pale yellow; slightly acid; transparent; 1.016; urea gr. vj to ℥j; no albumin; no sugar; and nothing abnormal microscopically.

May 5th.—The estivo-autumnal parasite was reported to be present by the pathologist.

May 8th.—The examination of the blood showed:—

Red cells	1,500,000
White	5,600
Hemoglobin	20%

As the boy had previously lived in a malarious region and was now residing in a locality noted for its large percentage of malarial diseases, I was convinced that the patient had chronic

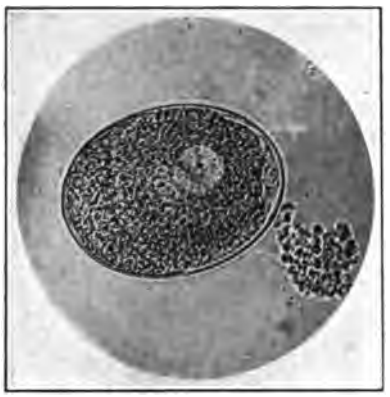


FIG. 1. UNICELLULAR OVUM.

FIG. 2 SEGMENTING OVUM.

FIG. 3. SEGMENTING OVUM, MULBERRY FORM.

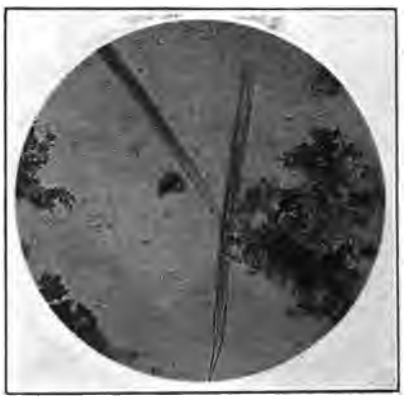


FIG. 4. ARTIFICIALLY HATCHED LARVÆ.

The accompanying plates show the ova and artificially hatched larvæ of *uncinaria Americana* prepared and kindly furnished by Dr. Carroll.

malarial intoxication, with secondary anemia, in spite of the absence of an enlarged spleen, and treated him accordingly.

Nutritious food, such as the best meats, fowl, eggs, and vege-

tables, was allowed. Eighteen grains of quinin sulphate was given daily for eleven days, during which the boy improved in appearance and felt stronger and his appetite was better.

Dr. James Carroll, of the United States Army, who examined the blood and feces reports under date of May 13th, said: "I find that it (stool) is loaded with ova of the uncinaria; there are thousands of them present and several may be seen in almost every field. They are beautifully typical in appearance, some containing segmenting ova and others young embryos."

The result of the blood examination today is as follows:

Hemoglobin	20%
Leukocytes	2,920
Red blood cells.....	2,181,600

The *uncinaria Americana* having been found, I was led to believe the anemia was due to uncinariasis rather than to malarial intoxication and directed the treatment applicable to the former to be carried out.

May 15th he was given a light supper and a tablespoonful of Epsom salts at bedtime. At seven o'clock the next morning the salts was repeated and an hour later 10 grains of thymol every hour and a half was given until 30 grains had been taken. An hour later another dose of salts was given. Food was withheld until 5 P.M. on this day. This treatment was repeated May 26th, June 5th and 12th as parasites and ova were present in the feces.

On May 29th he was given a solution of iron and manganese and continues to take it.

History during course of treatment:—During the first week he complained of feeling tired and weak and of headache. These symptoms have gradually disappeared and he now plays in the yard and waits on the younger children in the ward. His temperature remained normal and there has been an absence of sweats, nausea or vomiting and pain in abdomen; the constipation persisted. He now eats very heartily and enjoys his food. The pulse has improved. The murmur is still heard, but is not so intense. The skin is not so yellow and pasty. The tongue and lips are now pink.

Under date of June 13th, Prof. Ch. Wardell Stiles, Chief of Division of Zoology of the Public Health and Marine Hospital Service, writes: "Referring to your case of hookworm disease at

Children's Hospital, I would say that, in the various specimens of stools, which you have sent us, we have repeatedly found numerous eggs. The eggs vary from 60 to 70μ in length by 41 to 46μ in breadth, thus agreeing with *uncinaria Americana.* We have succeeded in developing from them the embryo and later the socalled encysted larvæ."

NOTE.—June 19th worms and ova were present in the stools and he was given thymol July 3d. Blood examination:—

Hemoglobin, ...	40-50 per cent.
Red blood cells	4,400,000
Leukocytes	9,100

July 5th and 6th worms and ova present. Thymol given. July 14th and 21st Dr. Stiles reported that the stools contained neither worms nor ova. He was discharged cured July 23, 1905, and has not been back for treatment, so it is presumable that the cure is permanent at this date, March 30, 1906.

DISCUSSION.

DR. ACKER.—I saw this case with Dr. Adams in the hospital, and the diagnosis was not at all clear. There were some indications of malaria, and yet the anemia was not that usually seen in that disease. The spleen and liver were both enlarged. Then it was suggested that the stools be examined, and the finding of the hook worm of course explained the nature of the disease.

DR. JACOBI.—Was any blood found in the passages?

DR. ADAMS.—None at all.

DR. GRAHAM.—I would like to ask Dr. Adams whether this parasite is supposed to inhabit any portion of the intestinal tract; whether it is found high up in the small bowel, or does it affect the large bowel. I would also like to ask whether irrigation of the colon and restriction of the diet plays any part in the treatment since it is an important part of the treatment for tape worm. I think removal of the head of the tape worm often fails simply because we do not irrigate sufficiently well to remove it.

DR. ADAMS.—The parasite inhabits the upper part of the small intestine. As to the treatment, the plan followed was, to give the patient a dose of Epsom salts at night, a second dose in the morning, and then 30 grains of thymol, given in 10 grain doses at hourly intervals. This was followed by salts again. A great many worms were passed in the stools, but the worms are difficult to find unless some parasiticide is given: the eggs only are found in the ordinary stools.

COSTAL SYNOSTOSIS ABOUT DRAINAGE TUBES IN EMPYEMA

BY FRANCIS HUBER, M.D.,
New York.

Simple incision with drainage, by means of rubber tubing, single or double, is usually all that is required in the operative treatment of acute empyema in children. This simple procedure, in the general run of cases, answers the purpose of drainage and permits the escape of even large masses of fibrin, etc. The cure in empyema is accomplished principally by the expansion of the lung (which has been compressed by the exudation of fibrin and pus), the chest wall falling in to a greater or less extent, depending upon the elasticity of the ribs. The displaced organs resume a more normal position as soon as the pressure of the fluid is taken away. The diaphragm, no longer restrained in its movements, begins to move with greater freedom and performs its proper functions.

Whatever interferes with the complete expansion of the lung tends to prolong the healing of the abscess cavity. Amongst these factors the principal are, too small an opening, the presence of firm pleural thickenings, deficient development of the thoracic muscles, or defective breathing, as is the case in nasopharyngeal obstructions caused by lymphoid hypertrophies of Waldeyer's tonsillar ring and finally close approximation of the ribs.

Under such circumstances and conditions, the prolonged retention of the drainage tube causes an erosion and absorption of the periosteum and bone in the immediate neighborhood. When the periosteum is irritated, its osteoplastic function is stimulated and new bone is thrown out from the osteal surface. Where the periosteum is destroyed, such changes cannot take place.

To digress for a moment, in the post-operative management of a case of empyema, too much stress cannot be attached to early pulmonary gymnastics tending to promote expansion of the lungs, as blowing soap bubbles, trumpets, the forcing of fluids from one bottle to another as suggested by Dr. W. B. James. Dumb-bells of proper weight may also be used. The patients ought to be encouraged to get out of bed as early as possible and be sent out into the fresh air. Infants should be taken up frequently and carried

about; when the weather is favorable they should be taken outdoors.

To return, a few weeks after simple incision, the probe frequently reveals "bare bone" at the point where the tube has been in contact with the rib. This must not be looked upon as necrosis,

SPECIMEN NO. I. EXTERNAL SURFACE.

SPECIMEN NO. I. PLEURAL SURFACE.

for if the abscess cavity is quickly obliterated, the sinus heals without any trouble. In cases continuing for six weeks and longer, more or less new bone is thrown on either side, above and below.

In the chronic cases, bony bridges, more or less complete, are formed, leading to a synostosis, as shown in the specimens pre-

sented herewith. The photographs were taken by Dr. I. Hirsch, radiographist to the Beth-Israel Hospital, to whom I herewith acknowledge my thanks and indebtedness.

As the prints show the salient points, no detailed description is required.

The following, in brief, is the history of the patient from whom the specimens were obtained: Perry C., three years old, was operated upon for the first time in August, 1903. May 15, 1904, he was admitted to the Children's Service, Beth-Israel Hospital, a pleural sinus still persisting and discharging a small amount of pus. May 23d, under chloroform narcosis, the writer removed the two ribs shown as Specimen No. 1.

SPECIMEN NO. 2.

As the pleural sinus was opened up a small amount of pus was found in a localized pocket. A number of firm adhesions between the parietal and visceral pleura were divided and the lung freed. In a few months' time the wound and sinus were fully healed. His general and local conditions were satisfactory for months, then the old sinus reopened and began to discharge again. At first the secretion was serous, later on it became purulent. A drainage tube was again inserted and weak solutions of iodin were injected. As there was no tendency to closure, the family finally consented to further operation.

March 30, 1905, under chloroform anesthesia, Specimen No. 2, consisting of portions of the seventh and eighth ribs, was removed from just below the angle of the scapula on the right side. Some

additional sections were taken away from the seventh rib upwards and forwards. The thickened pleura was also excised and the wound packed. At present there is a small granulating surface left.

In operations for the cure of pleural sinuses in chronic empyema, it is not uncommon to find the periosteum absent and the ribs corresponding to or in juxtaposition with the tube eroded and bare. Osteophytes may be thrown out and in advanced cases the ribs may be joined by more or less extensive bridges of bone.

When the tube enters the pleural cavity at a right angle to the chest wall, a circular opening corresponding to the calibre of the drain exists, the canal being completed by new formed bone on either side. If, on the other hand, the tube enters the pleura at an acute angle, a corresponding groove or channel would be formed in the new bone and the adjoining portion of the rib.

We have already referred to the fact, that irritation of the periosteum by a foreign body as a drain, will stimulate its osteoplastic functions and lead to the formation of new bone.

There is still another possible explanation. Whenever the ribs are closely approximated, in performing the operation by simple incision, there is danger of injuring the periosteum of the ribs. If cut or torn the edges are everted, new bone is thrown out from the osteal surface, and osteophytes and bony bridges result.

NOTE.—Since the above report, 3 more cases of chronic pleural fistula have been operated upon by the writer and similar bony deposits and bridges were removed by operation.

DISCUSSION.

DR. GRAHAM.—I have been very much interested in this last paper because I have always advocated and taught that in young children, children under two years of age, it was not best to do immediate resection of the rib, but to insert as large a drainage tube as possible. I have always considered that in such cases the elasticity of the chest wall allowed the abscess cavity to contract and that resection was unnecessary. If further studies show that the insertion of a tube, by its pressure, is apt to produce the changes shown in Dr. Huber's specimen I shall have to change my teaching and take the chances of a quicker recovery of the patient by the better drainage. If the experience of the other

members of the Society should confirm that of Dr. Huber it will make it advisable to do a prompt resection.

DR. KOPLIK.—I have some specimens at home, 2 cases exactly like these of Dr. Huber's, taken from cases of *resection.* As to Dr. Graham's inquiry, I have quite a large statistical record of empyema, and recently, in a paper read at one of the local societies, I collected all the cases of empyema in infants treated by simple incision and drainage tube, and found that the number of recoveries was just as large as in an equal number of cases in which I had resected a portion of one or two ribs, so that I am inclined to think that in young children and infants it is best to make the simple incision and drain.

You must not forget that empyema is a pleurisy with adhesions as well as thickening. In some cases where the pleura is simply inflamed, the lung expands after a simple incision and the child gets well. In other cases, whether you incise or resect, the patient does not get well because the lung cannot expand. Recovery in empyema is influenced a great deal by the pathological condition of the pleura, which in many cases is much thickened and prevents expansion of the lung. In such cases nothing short of even repeated resection will cure the case.

DR. JACOBI.—One remark of Dr. Huber's, if I understood him properly, surprised me a little. If I am not mistaken, he said that the presence of the drainage tube would set up an irritation by pressure and that was the cause of this bony communication between the two ribs, this bone proliferation. Was that right?

DR. HUBER.—Not quite.

DR. JACOBI.—What relation then is there between the formation of the bone and the drainage tube?

DR. HUBER.—The long-continued presence of the tube will cause an irritation of the periosteum, and the bone beneath the periosteum on either side gives rise to the new bone formation from the irritation produced by the presence of the tube.

DR. JACOBI.—That would explain some of the cases, but we must take into consideration that there would be some absorption of bone if there be pressure. The new bone formation must be the result of growth from the perhaps somewhat irritated periosteum, and that is why we retain the periosteum in all bone operations, and I can well imagine that a spicula of bone irritating the periosteum will influence it in throwing out more callus, but as a rule I believe we should rely upon the normal action of the periosteum alone. I remember a case of a young man thirty years ago, who was taken sick in Vienna with empyema, was incised by Sohroetter, and got apparently well, began to travel again and becoming sick once more he went into the clinic of Heinecke, in Erlangen, who resected part of two ribs. Half a year later he

presented himself at home with empyema. There was a great deal of offensive pus in the pleural cavity, which was removed in part by puncturing, but it was no use to think of anything else except resection of a large part of the bony thorax. There were bony arches between three of the ribs. I remember that case because it was one in which I performed a very large resection; it was a very difficult and bloody operation, after which the man recovered permanently. In that case I had no doubt that there was something more than the unusual irritation of the periosteum. Pressure alone will rather absorb than irritate.

DR. HUBER.—I did not intend to give the impression that all cases of acute empyema in children should be treated by incision. The principal feature in the cure is expansion of the lung. If the muscles are weak, respiration imperfect, or if there be thickening of the pleura, the drainage tube has to be retained a long time, and it is in these cases that the bony formation occurs. Ordinarily, where the tube is retained for several weeks, we find that in two to three weeks there is an area absorption of the ribs in contact with the tube; if the sinus heals there is no further trouble; if the tube has to be worn much longer the everted periosteum will throw out new bone tissue.

DR. KERLEY.—In my personal experience I always prefer resection of the rib. I think the surgeons all take that view of it and that the results of such treatment are more satisfactory.

DR. HUBER.—Most of the cases we see in the hospital in the lower eastern section of New York are such that it is imperative to operate quickly and with as little shock as possible; often there is edema of the other lung and it would be dangerous, perhaps, to submit the child to an anesthetic and more prolonged operation.

CASE OF PORENCEPHALUS.

BY G. N. ACKER, M.D.,
Washington, D. C.

L. P., eleven years old, female, colored. Her mother died of typhoid fever. No history of the father. No other children. The child was breast-fed for three weeks, then fed on cow's milk. None of the diseases of childhood. She has never been able to walk or even stand. Appetite has been good, but bowels constipated.

About February 1, 1905, a bloody discharge was noticed coming from the vulva and a protrusion from the same. This condition has been very painful and the child has been unable to sleep on account of it.

Admitted February 10th to Children's Hospital. She was always in a recumbent posture; thighs and legs flexed. She was poorly nourished. Her chest was very long. Appetite good. Called for milk and meat. Abdomen slightly distended. Both lower and upper extremities much deformed. She never was able to see. Considerable exophthalmos and nystagmus. Sense of hearing poorly developed. She understood when spoken to and would answer questions. Power of speech, however, slight. Power of motion absent, except in upper extremities. The following history of the case was kindly furnished by Dr. Julia R. Hall, under whose care she was for some years.

Lizzie was born December 23, 1893. She had the appearance of a normal child at three weeks of age, when she first came under my care. Her mother was a very young woman. A domestic, but not up to the standard mentally. Father was unknown to me. The mother died when Lizzie was a little more than a year old, with typhoid. The child had large, beautiful dark eyes, and it was some weeks before we discovered that she was blind.

Slight deformity of the bones became noticeable at beginning of the second year, which continued to grow more as she grew older. She was never able to sit alone. Never had any use of arms or legs, more than to hold a rattle in one hand. After the third year she would respond by a laugh or crooning noise when talked to, or sung to, but could not talk.

When five years old, she could say "milk," when she was hungry, and at another time she would say "Mamma," or "piece a bread," which was about the extent of her vocabulary.

FIG. 1. RIGHT SIDE OF BRAIN.

FIG. 2. LEFT SIDE OF BRAIN.

She had the sense of smell and taste. After she was taken off the milk diet we found that certain articles of diet she would take and seemed to relish, others she would refuse. She never chewed,

although she had a good full set of teeth. She took her food by sucking as she did her nursing bottle. We used a food chopper to reduce whatever she ate to the proper consistency. She never had any of the diseases of children, although the other children in the house went through the whole list, including diphtheria, in the same room.

On admission to the ward she was so very restless that it was

FIG. 3. VIEW OF BRAIN FROM ABOVE.

found necessary to put her under the influence of opiates and continue the administration during the rest of her life.

On the 14th, Dr. J. Ford Thompson made an examination and found a gangrenous condition of the urethra, extending almost to the bladder. Examination of the bladder was negative. The gangrenous portion was excised. The temperature ranged from 97.8 to 104° F., and was irregular. She died on the 15th. She chewed her food while in hospital.

The postmortem examination was made by Dr. D. S. Lamb: Body extremely emaciated. Upper border of right ear showed ulceration. Bones of limbs much curved. Brain showed nearly

symmetrical porencephalous cysts involving the parietal lobes and parts of temporal and occipital, and extending to the lateral ventricles. Neck and spine not examined. The lungs showed some tubercles and some old adhesions. The heart was atrophied, weighing only 80 grams; much serum in pericardium. Bronchial and mediastinal glands enlarged and tubercular. Dirty liquid in peritoneal cavity, and coils of small intestine adherent together. Mesenteric glands formed a large tubercular mass. Stomach normal. Small intestine showed tuberculous ulcers, one of which had perforated, causing peritonitis. Tubercles on peritoneal surface of ulcers. Some small tubercles in liver. Spleen lobulated and tubercular. Pancreas normal. Right kidney normal; small cyst in left. Bladder empty.

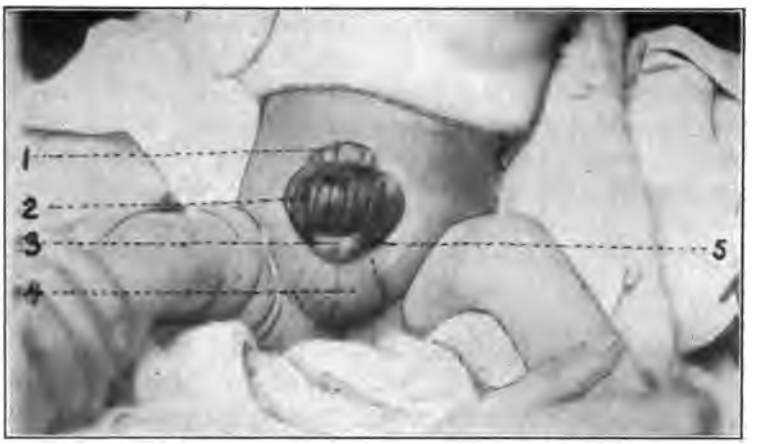
The condition of the brain explains the absence of vision. The question as to muscular action is not so clear, as some of the parietal region and all that related to motion was present. The centre of speech was evidently not affected, as the child could speak, though only a few words. Hearing, taste and smell were normal.

CASE OF CONGENITAL EXSTROPHY OF THE BLADDER.

BY A. C. COTTON, M.D.,
Chicago.

Baby T., two months of age, bottle fed and somewhat emaciated was brought to my clinic by Dr. Lang.

The mother is German, twenty-one years of age, one of a family of fifteen, all without anomaly; primipara; two years ago she is said to have had cystitis, apparently ovaritis from descrip-



EXSTROPHY OF BLADDER.

1. Umbilical Cicatrix. 2. Bladder wall. 3. Rudimentary penis. 4. Scrotum. 5. Inguinal hernia.

tion of symptoms. She was in the county hospital for four months, at the end of which time she was discharged as cured without operation. Soon after she married. The first two months after marriage and while pregnant she worked in a steam laundry where a rapidly moving board continuously struck her abdomen. She began to have regular labor pains and was obliged to stop work. The pain subsided to a degree after twenty-four hours but some discomfort was complained of during the remainder of pregnancy. She was delivered at full term without incident by a midwife. The father, twenty-one years of age, a barber, is in

good health, has three brothers and one sister, all well and normal. His father died of phthisis, mother still living.

The baby shows the following anomalies: The umbilicus is represented by a red cicatrix and beneath it appears a red vascular tumor the size of a small orange, occupying the central portion of the abdomen from the umbilicus to the root of the penis. Upon lifting the protruding mass of extroverted bladder we can see at its lower aspect the orifices of the ureters discharging urine in drops. Below it we have an organ, or more correctly one-half of it, suggesting the lower half of the penis as if the top part were sliced off. It shows a linear central depression, the urethra probably. Below this is the scrotum without contents. The bones are wanting at the pubic symphysis. At both external abdominal rings we find a small body apparently a testicle above each one of which is a hernial tumor.

NOTE.—The babe was kept under observation with a view to reparative surgery, but succumbed to an attack of enterocolitis before his strength would warrant an operation. Autopsy was not allowed.

MULTIPLE ABDOMINAL TUMORS, DUE TO CYSTIC KIDNEYS, WITH DOUBLE HYDRONEPHROSIS AND GREAT DILATATION OF THE URETERS, HYPERTROPHY OF THE BLADDER, ETC., IN A CHILD DYING AT FOURTEEN MONTHS.

BY L. EMMETT HOLT, M.D.,

Professor of Pediatrics in the College of Physicians and Surgeons (Columbia University), New York.

The family history in this case was entirely negative. There was but one other child and that healthy; both parents were living and well; and no history of hereditary disease in the family.

The patient was a full-term child, born after a natural labor. The mother stated that it was remarked by the doctor, at birth, that the abdomen was very large, "like an alderman's." The child was fed artificially after the first week, and gained properly. No further attention was paid to the abdomen, though it was said that the "lumps" were always present.

At the age of three months, the family became concerned as the masses seemed larger and, shortly after this, the child was taken to one of the large hospitals, where a diagnosis of sarcoma of the kidney was said to have been made and operation advised. This was refused and the child was taken home. A few weeks later diarrhea developed, and for this he was admitted to the Babies' Hospital on August 4, 1904.

On admission the child, now six months old, was well developed; weight, 14 pounds, 7 ounces; he was pale and rather cachetic in appearance.

The following is the record of the examination made at that time: Abdomen very prominent, distended, tense, and for the greater part, tympanitic; after irrigation, no rigidity and no tenderness. Three large abnormal masses were felt: one in the left lumbar region, somewhat irregular in outline, about the size of an orange, apparently not connected with the spleen, somewhat movable; a second tumor in the right lumbar region, not quite so large and not quite so prominent; a third tumor, smooth and round in outline, a little smaller than an orange, in the right iliac region and lying to the left of this a small mass about the size of

a marble, "apparently a gland." The lower border of the spleen was palpable, but did not come below the ribs; the lower border of the liver could not be made out distinctly. Except for a moderate phimosis, genitals normal.

The child was under observation with a moderate attack of enterocolitis until August 22d, when he was discharged, no change in the abdominal condition having taken place, and with no symptoms except those of the intestinal condition; no temperature after the second day. There was nothing else of importance in the physical examination, which was fully recorded.

October 10, 1904, he was readmitted. The history stated that, after discharge in August, he had improved and had gained in weight until one week before, when some digestive symptoms developed, looseness of the bowels, loss of appetite, etc., in consequence of which he was brought again to the hospital. The mother had observed that the abdominal tumors had increased in size, but not greatly.

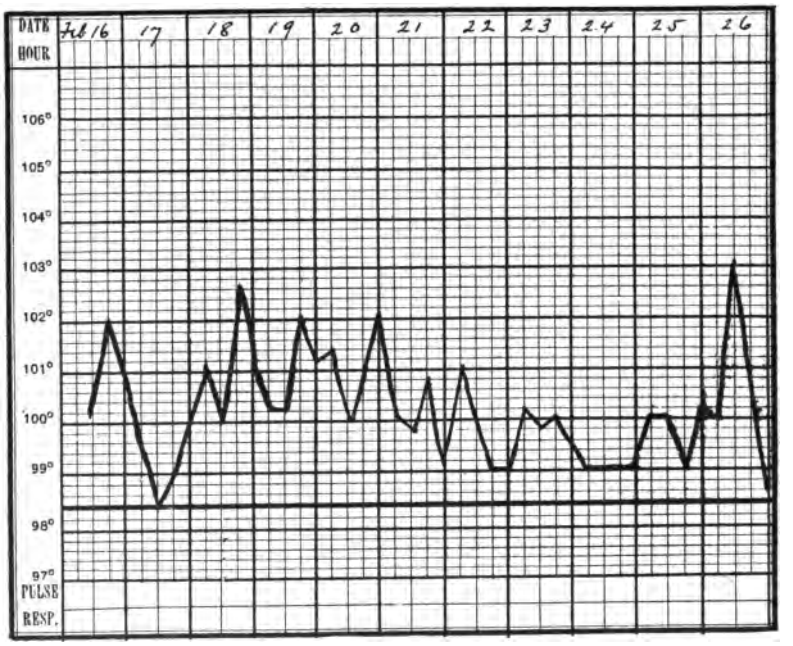
His general condition was not good; he was pale, poorly nourished and weighed only a few ounces more than at the time of his discharge in August. There was no marked change in the abdominal condition. The position of the tumors was nearly the same; the one in the right iliac region was nearer the median line; the small gland-like tumor at its left side was nearly an inch in diameter. The lumbar tumors seemed only slightly larger than at the previous examination. See Figure I., page 187.

The child remained in the hospital for eighteen days, during which time his digestion improved very much and he gained one-half pound in weight. He had no fever. The urine was collected for twenty-four hours; 9 ounces were passed; examination showed it to be a pale amber color, slightly cloudy; specific gravity, 1004; no albumin; crystals of uric acid; no casts, and nothing else abnormal. There was now a leukocytosis of 10,900. His only symptoms were intestinal, and he was discharged greatly improved, though not quite well.

January 10, 1905, he was readmitted for observation. He was now eleven months old; his weight 18 pounds; well developed, and in general appearance much improved, in fact, a very healthy looking child. He has been very well since his discharge in October, gaining steadily in weight. The mother had not observed any change in the condition of the abdomen and now, as before,

on account of his excellent general health, she refused to allow an exploratory incision or any other operative interference.

Examination showed no marked change in the situation, size or appearance of the abdominal masses, except that the lumbar tumors were more elongated vertically and not so globular as they were previously. They were sharply outlined, but of irregular shape; firm, hard and, as before, movable only to a limited extent. Nothing new was learned during this period of observation.



TEMPERATURE CURVE DURING THE LAST ILLNESS.

There was no fever except during a mild intercurrent attack of German measles. The digestion was good; the bowels regular; no abdominal pain or tenderness present, and no fluid made out in the abdominal cavity. The percussion now, as always, was tympanitic except over the tumors. Discharged January 28th.

February 16, 1905, he was readmitted with fever and vomiting which had existed for three days and had come on rather acutely. The vomiting was repeated five or six times a day; the bowels had been constipated; some abdominal pain seemed to be present, as he cried when handled or moved. The abdominal tumors were

not essentially changed except that the one in the left lumbar region seemed slightly larger than a few weeks before. His weight was 18¾ pounds, and though well nourished, he looked seriously ill.

From this time until his death ten days later, his temperature continued elevated (see chart), although it was never high. He took but little food, vomited occasionally and grew steadily weaker. The abdominal tenderness was unmistakable and was particularly marked upon the left side.

On February 21st the urine was of a pale yellow color; alkaline reaction; somewhat cloudy; containing a trace of albumin and a few hyaline casts. There were numerous pus cells and a few red blood cells. There was at this time a leukocytosis of 21,000. Three days later a specimen was drawn with a catheter for examination and showed a much larger quantity of albumin, a greatly increased amount of pus and numerous cocci. The leukocytosis was now 23,000.

During the next few days the child failed rapidly; he became dull and apathetic; the vomiting returned; the circulation became very poor; he refused all food and finally passed into a condition of collapse, in which he died on February 26th.

The outline of the tumors given in the accompanying sketch (Figure I.) represents the relation present during the greater part of the time the child was under observation.

Autopsy.—The brain was not examined. The heart, lungs and bronchial lymph nodes were normal. On opening the abdomen, the cavity was found to be nearly filled with two large irregular masses, the principal tumors felt during life. These proved to be two cystic kidneys of great size with an enormous double hydronephrosis.

The place of the left kidney was taken by a large lobulated cystic tumor, whose outline bore little resemblance to a kidney; it measured 16 cm. in length, and each lobule appeared like a cyst. The pelvis was funnel-shaped, measuring 5x9 cm. There were in places areas of renal tissue over the convexity of the tumor, but the cortex was everywhere a mere shell; in many places no trace whatever of renal tissue was visible. The ureter was larger than the colon and measured 7 cm. in circumference at its entrance into the bladder; it was elongated, tortuous and convoluted, closely resembling a much distended small intestine. The small tumor, so

constantly felt in the hypogastric region, was formed by a convolution of the ureter.

The right kidney was similar to the left, although the process

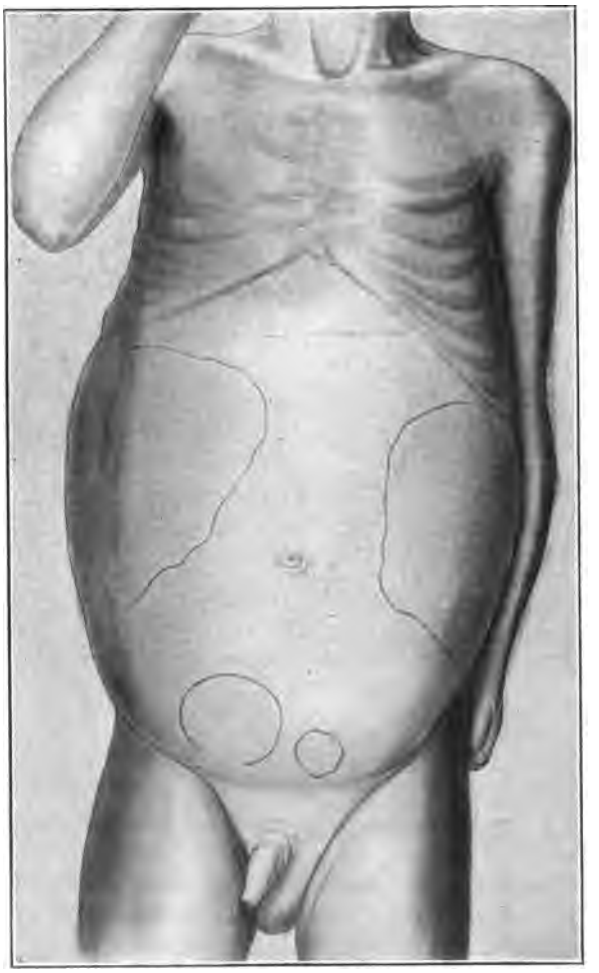


FIG. I. OUTLINE OF THE ABDOMINAL TUMORS AS THEY APPEARED DURING LIFE (AFTER A PHOTOGRAPH).

had not advanced so far; it was 12 cm. long. A thin cortex of renal tissue, nowhere more than 2 or 3 mm. in thickness, was present over the greater part of the convexity of the tumor. The pelvis of this kidney measured 3 by 6 cm. The ureter was about

two-thirds the size of that of the left side and convoluted in a similar way.

The bladder was very prominent, and it was this which formed the ilio-hypogastric tumor; it measured 6 by 8 cm. Its walls were greatly hypertrophied, measuring more than 1 cm. in thickness. The mucous membrane was pale, but not inflamed. There was no obstruction in the ureters, at the meatus, nor in the urethra. The general outline of the tumors, the bladder and the ureters are well shown in Figure II.

FIG. II. DOUBLE HYDRONEPHROSIS, DILATED URETERS, ETC.

The mesenteric and retro-peritoneal lymph nodes were enlarged, but not cheesy. There was no peritonitis. The contents of the left tumor was a turbid fluid, that of the right was clear.

The stomach, intestines, liver and spleen were normal.

Only one similar case has come under my notice ("Diseases of Infancy and Childhood," third edition, page 657). It occurred in a child one month old dying of marasmus. There was in this also the greatly hypertrophied bladder, and the dilated, tortuous ureters; the condition in the kidneys, however, was not so far advanced, probably on account of the age of the patient. There

was the same absence of obstruction in the ureters or urethra. The advanced changes present at one month made it evident that the condition was congenital.

The pathological conditions found in these two cases would appear to point strongly to some obstruction to the outflow of urine as an etiological factor, and yet none could be found in either case. In the patient whose case is here reported, the urine was passed without the slightest difficulty throughout its life; there was never any retention, and catheterization was done repeatedly without difficulty during life. No narrowing of the urinary tract was discovered after death. The condition present evidently was one which dated back to intrauterine life, and must have occurred a considerable time before birth, as the tumors were evident when the child was born.

I can find on record but two or three similar cases, none so marked as in the two here referred to. They are usually classed as primary hydronephrosis of congenital origin, but obscure in their etiology.

The clinical course in this case seems quite extraordinary. That a child should not only live to the age of fourteen months, but that he should thrive to all appearance in a normal way with such an extensive deformity, seems most surprising. In one reported case a child lived to the age of four years with double hydronephrosis, but not so marked as in the case here described. In my own patient there was apparently enough renal tissue left to carry on the functions of the organ. The cause of death seemed to be infection of the left kidney—the conversion of the hydronephrosis into a pyonephrosis.

The prolonged course of hydronephrosis and the absence of both local and constitutional symptoms is in striking contrast to other tumors of the kidney and in fact of most abdominal tumors present in young children. There was really very little change discernible in the conditions present in this patient from the time he came under observation in August until the close of life in February; and no changes of importance were present in the urine except during the last two weeks.

The diagnosis during life presented great difficulties. The first one made of a sarcoma was shown to be erroneous by the nearly stationary character of the tumor and the normal growth and development of the child. Two or three physicians who saw the

case were positive at first that tubercular peritonitis existed. The diagnosis, however, was finally narrowed down to a cystic condition of the kidneys. The continued absence up to the last two weeks of any evidence in the urine of serious trouble seemed, however, to be against it. The final appearance of albumin, casts and pus led us to make the correct diagnosis so far as kidneys were concerned, but the two tumors in the lower part of the abdomen remained an enigma which was not solved until the autopsy, although catheterization on the night of the child's death made it evident that the larger tumor was the hypertrophied bladder. It is to be noted that in the early months this bladder tumor was not in the median line, but rather in the iliac fossa.

In view of the autopsy findings, operation would, of course, have been out of the question. In cases where the disease is unaltered it should always be advised, as infection of the hydronephrosis is almost certain to occur sooner or later.

The diagnosis is to be made chiefly from sarcoma of the kidney, from which it is distinguished by its very slow growth and the absence of constitutional symptoms. In a female child an ovarian cyst might be mistaken for hydronephrosis.

The etiology of congenital hydronephrosis is very obscure. It is apparently a primary condition as neither calculus nor any other causes of obstruction are present. It is important in this instance that during life there were no symptoms of retention of urine or of difficult micturition.

14 West Fifty-fifth Street, New York.

DISCUSSION.

DR. BLACKADER.—What were the clinical symptoms, were there any referable to the bladder and urinary organs?

DR. HOLT.—Absolutely none. The child came in for observation mainly, but repeated examinations showed no blood and no pus until just before death. These tumors were fully four times their present size when first taken out.

DR. HUBER.—In regard to Dr. Holt's case, I would state that Dr. Peck, Junior Surgeon at Roosevelt Hospital, had diagnosed the case as one of double congenital hydronephrosis. I know of a case of congenital hydronephrosis operated upon at Gouverneur Hospital by Dr. H. M. Silver; the tumor was drained first and then, when the child had improved, the kidney was removed.

DR. HOLT.—That is the history of most of the reported cases.

EXAMINATIONS OF THE URINE OF INFANTS.

BY HENRY DWIGHT CHAPIN, M.D.,
New York.

The difficulties in connection with a study of the urine of infants are shown by the vague and conflicting accounts given by the various authors in regard to this excretion. The importance of this study, both in health and disease, is obvious to the practicing physician. The rapid metabolism occurring at this time of life and the vulnerability of the kidneys will occur to every one. A careful examination of the urine in various conditions is presented in the following series of cases from the author's service at the babies' wards of the New York Post-Graduate Hospital. The first series includes 86 cases in which some disturbance of the gastrointestinal tract was present. No attempt was made to classify these cases and they include simple indigestion, fermentative diarrheas, catarrhal inflammation and marasmus. In a large number the condition was not severe and such cases were purposely included in the list. Albumin was present in 75 cases in this series of 86. Its presence was noted as follows: trace 29; faint trace 31; heavy trace 15. Casts were present in 37 cases, noted as hyaline, granular, epithelial and mucus. There were sixteen deaths in the series, and of these fourteen had albumin present and ten both albumin and casts. In 32 cases an examination for indican was made and found present in 22 of the cases. The amount was estimated as follows: trace 4; faint trace 1; heavy trace 17.

A series of 57 cases of pulmonary diseases, such as severe bronchitis, pleurisy and pneumonia, gave the following results: 49 had albumin in the urine, thus noted; trace 13; faint trace 30; heavy trace 6. Thirty-two cases had casts present, either hyaline, granular, epithelial or mucus. Of the seventeen deaths in this series, fifteen had albumin present and ten both albumin and casts. An examination for indican in 23 specimens showed its presence in 16 cases. Trace 2; faint trace 2; heavy trace 12.

In 45 cases of general illness, other than pulmonary and gastrointestinal, albumin was present in 31 cases. Trace 9; faint trace 11; heavy trace 11.

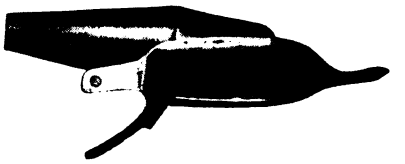
In 11 cases of cerebrospinal meningitis, 9 showed heavy traces of albumin and casts.

It is evident that any disturbance of the bodily functions during infancy will often be accompanied by the presence of albumin and casts in the urine. What significance does this condition present? Can actual renal disease be considered to exist when traces of albumin and a few casts are found? or is there simply an irritation of the renal tubules accompanying a slight congestion and having no special significance? To the writer's mind a study of the cases here reported favor the latter view. Koplik in a study of 25 consecutive cases of gastroenteritis found that all but 4 showed a more or less severe involvement of the kidney. In all of these cases there was albuminuria and the majority of them showed the presence of casts. This author further says that in view of the peculiar physical signs and the rapid improvement of an almost complete suppression, without leaving behind any appreciable lesion of the kidney as evinced by albumin or casts in the urine, it is seen we are not dealing with a nephritis in the ordinary, but in a special sense. As in these cases there is usually a great loss of fluid from the system, the toxins circulating in the different organs are thus placed in contact with the delicate cell structures in concentrated form. As soon as the water taken from the system is partially supplied, these poisons are washed from the organs and the latter have an opportunity to resume their functions and are restored to normal. The moral is not to employ irritating antiseptics in the treatment of intestinal diseases and to give a full and free supply of water.

It would seem that we are justified in concluding that the urine of infants may contain traces of albumin and even casts without any very grave results. Martin Ruge states that hyaline and granular casts may be found in the urine of the newborn. Even when actual congestion or parenchymatous inflammation exists for quite a long time, it may be remembered that in early age the kidney possesses a wide power of regeneration. In the ARCHIVES OF PEDIATRICS (June, 1905), Dr. Edwards reports cases where the growing kidney presented urine containing both albumin and epithelial cells, and where he was unable to say that either the cardiovascular or the renal apparatus was organically at fault. These cases have been later examined in middle life and found without demonstrable renal or cardiac diseases.

The exceedingly fine tests now often employed in examining

for albumin must be noted as one explanation of its frequent discovery. As small amounts of nucleoproteid are always present in urine, probably derived from the disintegration of the epithelial cells from some part of the urinary tract, such as the ureter or bladder, fine traces of albumin may come from such a source.



INFANT'S URINAL.

The difficulty of procuring specimens of the infant's urine for examination accounts for much of the laxity in this respect. The usual methods of the sponge and cup are unsatisfactory, and the passage of a catheter upon the young infant is difficult and not

URINAL IN POSITION.

without danger. To obviate the trouble and make feasible the routine examination of the infant's urine, the writer has devised a small urinal that has proven an easy and safe method of collecting the urine for examination. It consists of a circular opening ending in a funnel that fits into the collecting vessel. Two sizes have been found necessary, small and large, designated respectively as No. 1 and No. 2, for infants under and over one

year. The urinal is fixed in place by putting the large opening around the vulva in the female and over the parts in the male with the funnel pointed downward. Tapes are put through the openings in the arms and fixed by tying around the abdomen and both groins. To fix more firmly in place, strips of adhesive plaster may be pasted over the arms. The end of the funnel is placed in a collecting bottle which is kept in position by the diaper. If the baby is very restless, a cork may be put in the end of the funnel and the bottle dispensed with, as enough will often be thus collected for examination.

After frequent changes of the model and two years trial at the Post-Graduate Hospital, the present form has proven satisfactory. The writer had hoped that the apparatus would enable one to collect the full amount passed in twenty-four hours, but this has not proven feasible without constant watching, and the movements of the baby make a little leakage unavoidable. In a number of cases of cerebrospinal meningitis, with coma, a special effort was made to collect the twenty-four hours' amount. A baby of nineteen months passed 18 ounces; one of two years passed 16 ounces, one of three years passed 16 ounces, and one of four years passed 20 ounces. All of these specimens had traces of albumin and casts and the urea varied from 1.7 per cent. to 2.7 per cent.

DISCUSSION.

DR. HOLT.—I would like to ask why the lower tube of this instrument is made so large. Could it not be made smaller so as to permit the attachment of a piece of ordinary rubber tubing?

DR. CHAPIN.—I tried that, but concluded it was not so good. With the short, broad tube fitting into a bottle, the napkin holds the entire apparatus in place, whereas with a long rubber tube running down between the legs it is difficult to keep it in position.

DR. EDSALL.—Does the apparatus cause any irritation, and can the child wear it for a week or ten days?

DR. CHAPIN.—I have had them worn for several days and they caused very little disturbance.

DR. EDSALL.—Is it absolutely closed?

DR. CHAPIN.—No; it may lose a few drops; it is not sufficiently accurate for metabolism investigations.

DR. CRANDALL.—As to the possibility of albumin and casts appearing in the urine of very young children, I can corroborate

that. I first examined for them in a child with supposed scarlet fever. It was an infant one week old, and there was a question of diagnosis. The child had a sore in its mouth, and Dr. Jacobi, who saw the case with us, thought that the rash was due to infection. An examination of the urine disclosed the presence of albumin and the casts, but in examining the urine of other healthy infants during the first week I have a few times obtained the same results.

DR. MORSE.—I have made it my habit to have the urine of every baby entering my service at the Infants' Hospital examined. I have not found albumin, by the heat and nitric acid tests, in as large a number of cases as Dr. Chapin reports. Of course, we find casts in only a small proportion of the cases in which there is albumin. It seems to me that the explanation of the presence of albumin and casts in acute diseases is the same as in adults; they are due to an acute degenerative nephritis, the result of the elimination of toxic substances through the kidneys. Generally, this means nothing of importance.

DR. MCCOLLOM.—I was much interested in what Dr. Chapin said about albumin and casts in children and the comparative insignificance of it in connection with acute diseases. A large number of children have albumin in the urine at any rate. I was very glad that Dr. Chapin brought out that point, for the child's kidney will recover much more quickly from such diseases as scarlet fever than will the adults.

DR. JACOBI.—On the other hand, the very fact that albumin will show itself in a number of cases where it means nothing or very little, makes it worth while to remember that nephritis is a very frequent disease in the very young,* very soon after birth, and through the whole course of infancy. Nephritis is frequent and pyelitis is not at all rare. Albumin then, may mean nephritis unless the condition disappears in a very few days, and a single negative result of the urine examination is not conclusive.

DR. JENNINGS.—What Dr. McCollom said in regard to the insignificance of albuminuria in children would seem to justify, and I think there ought to be, a decided distinction between acute degeneration of the kidney and nephritis. From the results and the symptomatology of the cases of albuminuria that are so frequent in acute diseases it seems to me the condition is not a nephritis. I am sorry Dr. Morse calls it even an acute degenerative nephritis. I think it is a degeneration and not an inflammation. Acute degeneration with albumin and casts will occur in the beginning of scarlet fever and pass away, but later on an actual nephritis may occur, yet these are two different things.

DR. GRIFFITH.—I would like to ask Dr. Chapin what tests he used in examining for albumin. My experience has been about

* *Cf. New York Medical Journal*, January, 1896.

like that of Dr. Morse, that I do not find it so frequently as Dr. Chapin seems to have done. I have been in the habit of using the ordinary heat and nitric acid tests.

As to the collecting of urine, we have been successful in the hospital with catheterization in infant girls, and with the use of a wide-mouthed bottle for infant boys. The apparatus which Dr. Chapin has exhibited seems to me, however, to promise to be an extremely useful one.

DR. KOPLIK.—In obtaining the urine of infants, I have never caused infection by using a metal catheter for females and a rubber catheter for males.

I have been surprised recently to find the frequent occurrence of very slight traces of albumin without formed elements in children suffering from gastric and intestinal diseases, but on the other hand that albumin with casts belonged to a distinct set of cases. Albumin itself is a frequent element in connection with disturbances of the stomach in children, and in slight amounts is of no serious import.

DR. CHAPIN.—The test I used was Esbach's, a very fine test, and its excessive fineness probably accounts for the larger proportion of cases in which albumin was found.

This apparatus, I should have said, fits the male baby as well as the female. I do not question the fact that in the hands of the expert, catheterization is all right, but it seems to me that if it is to be used as a routine practice, there are some dangers connected with it, and that examinations of urine will be more frequent and more thorough if the methods of collecting it can be simplified.

I was interested in what Dr. McCollom said about the different significance of the finding of albumin in children and in adults. I would not wish the Society to adopt the notion that nephritis is not grave and to be guarded against, but the question comes up in regard to diagnosis where we are not sure as to the trouble, and I am certain that I have seen the diagnosis of nephritis made when it turned out eventually that the albuminuria was only an incident and not the main thing.

DR. JACOBI.—I would like to know whether Dr. Chapin, in using Esbach's method, heats the urine.

DR. CHAPIN.—Yes.

DR. JACOBI.—And whether the diagnosis of albumosuria is excluded.

DR. CHAPIN.—Yes; I have not made the tests personally, but the work has been well done.

DR. JACOBI.—There is no better test for albumin than the oldest test—that of boiling with nitric or acetic acid; it is more conclusive than anything else and avoids confusion with albumosuria.

HODGKIN'S DISEASE WITH A MILKY NON-FATTY PLEURAL EFFUSION.

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The general question of the nature of Hodgkin's disease and of its relation to tuberculosis has been so frequently discussed in recent years that isolated instances of the disease are of little interest in this connection, excepting in so far as they aid in accumulating evidence. The general features in this case will be briefly mentioned for the latter purpose, since it was determined in this instance that the child's tissues were wholly free from gross or microscopic evidences of tuberculosis, and the injection of a number of glands into guinea-pigs produced no disease. The condition of the blood, more particularly the differential count of the leukocytes, is also worthy of passing notice, since this is a question that has been held to be of importance in diagnosis, but one upon which there has been difference of opinion. The general anasarca that occurred in the case is also of interest, because of the absence, both clinically and at postmortem, of evident cause for it, beyond the pressure of the generally enlarged glands. Universal dropsy is certainly explained with difficulty on the last mentioned grounds.

The feature of the case that aroused the chief interest, however, from the clinical as well as the pathological standpoint, was the non-fatty milky effusion that was twice withdrawn from the left pleural cavity, and that was found in this cavity postmortem. A considerable number of such effusions have been carefully studied, and probably some degree of milkiness of effusions resulting from substances other than fat is quite common; but the occurrence of such a condition is but infrequently commented upon, and it is indeed not mentioned in a number of somewhat elaborate recent papers on milky effusions. There is a rather general tend-

ency to describe all such effusions by the term chylous, even if a chemical examination is lacking, and in case even a small amount of fat is found by chemical or microchemical methods, the term chylous is frequently considered justified. That there are ready opportunities for error in such free use of this term is more or less generally known, but it becomes, I think, even more emphatically evident after an examination of the literature concerning this subject. The important facts shown by the literature on this point will be briefly referred to later after discussing some of the other interesting features in my case.

The history of the case was as follows:

The patient was a boy, aged twelve years. He was admitted to the Episcopal Hospital on February 22, 1905, and died there May 10, 1905. He was under the care of Dr. A. A. Stevens until April 1st; from that time until his death under my care.

His family history was of interest only in the fact that one sister had died of tuberculosis, and that his father was found to present scars on his neck, which he said were the results of operation in childhood; these suggested, of course, old tuberculous adenitis.

The patient's previous history included measles and whooping-cough in early childhood, without any known sequels; and he also had typhoid fever two years before admission, but otherwise had been well, until December, 1903, when a small lump appeared on the left side of the neck and rather rapidly increased in size. This was removed at the Jefferson Medical College Hospital in July, 1904. He rapidly recovered from the operation, but soon after his discharge from the hospital a small lump appeared near the operative scar, and this quickly increased in size, and at the same time a number of other smaller lumps were noted, anteriorly and posteriorly to the scar. His general health did not suffer appreciably until about three months before his admission to the Episcopal Hospital, when he had sudden sharp and rather severe pain in the abdomen, which continued for some time afterward, finally becoming intermittent, but persisting in the latter form until about two weeks before his admission. He had had pain in the chest and back also, though this was not severe. Throughout this period he had occasional attacks of mild diarrhea without known cause; there had been no other digestive disturbance. About two weeks before admission it was noted that his abdomen was increasing in size, and a few days later than this swelling of

the scrotum and prepuce had appeared. A short time before admission he began to have slight cough.

His condition on admission was briefly as follows: He appeared fairly well nourished, but had marked pallor, his face was puffy looking, and his expression dull. His pupils and eye movements were normal, the tongue coated, the throat clear. On the left side of the neck there was a scar about four inches long, and anterior and posterior to this scar were numerous large glands, the largest being one and one-half to two inches in diameter, while at least eight to ten more were fully an inch in diameter. Many other small glands could be felt on both sides of the neck, both in front of and behind the sternocleidomastoids, several of those on the right side being about an inch in size. There were a considerable number of enlarged glands in both axillæ, the largest, in the left axilla, reaching an inch and a half in diameter. In each groin there were also enlarged glands, though these were not more than half an inch in diameter. All these glands were apparently free from adhesions to the skin, and could be moved over one another and over the underlying tissues; all were of smooth and rounded surface, and felt elastic and rather soft, but not cystic; none of the glands was tender or caused pain. There was slight edema of the whole surface of the trunk, arms, and hands, and marked edema of the legs, scrotum, and prepuce.

The examination of the lungs was negative, excepting that there was rather prolonged and somewhat harsh inspiration on both sides and a few râles were found scattered over both sides. The heart dullness was of normal size and position, the apex beat in the fourth interspace slightly to the left of the nipple; there were no murmurs. The abdomen was prominent, the veins of the abdominal wall moderately distended, there was movable dullness in the flanks, but no succussion. Palpation of the liver and spleen was unsatisfactory because of the distention; no enlargement of either could be determined.

The urine showed: Specific gravity 1.020, acid reaction, a trace of albumin and a few hyaline casts. There was no sugar. The blood examination showed erythrocytes 4,272,000; white blood cells, 14,000; hemoglobin, 67 per cent.

Four days later the edema of the scrotum and prepuce had disappeared, but the legs and abdominal walls were very edematous, and there was a small amount of fluid in the left pleura. The leukocytes at that time were 13,600. The differential count

showed polymorphonuclears, 95.5 per cent.; large lymphocytes, 2.0 per cent.; small lymphocytes, 2.0 per cent; eosinophiles, 0.5 per cent. An X-ray picture of the chest was negative. A week later a decided increase of the fluid in the left chest was noted, the dullness extending to the fourth rib in the nipple line. The fluid in the abdomen was also increasing and all the enlarged glands had increased in size, particularly those in the right cervical region. Leukocyte count, 11,400. Differential inspection again showed that nearly all the white blood cells were polymorphonuclear. The apex beat of the heart was in the fifth interspace just to the left of the left border of the sternum. March 13th, 900 c.c. of fluid were removed from the left pleura. This fluid was so milky that when Dr. King, the resident physician, saw it appear, his first impression was that it was pus; when examined more closely, however, it had very much the appearance of a somewhat diluted and rather yellowish milk. Perhaps it may be more clearly described by the statement that it resembled whey that had been very imperfectly separated, and that still contained a considerable quantity of casein and fat. The specific gravity of the fluid was 1.012, the reaction slightly alkaline. When filtered it retained its previous appearance, and prolonged centrifugation was without apparent influence upon it. Microscopically it showed numerous lymphoid cells, an occasional polymorphonuclear cell, and vast numbers of very fine, highly refractive globules that had somewhat the appearance of the globules of a fine fat emulsion. There were no red blood corpuscles in it, and no bacteria were seen. Cultures from the fluid proved negative. The leukocytes at this time were 17,000, and again differential inspection showed that nearly all were polymorphonuclears.

After the aspiration the apex beat returned to about the normal position; a marked friction rub was heard over the whole of the right chest for several days after this. The enlarged glands continuously increased in size and others appeared; the edema constantly became more marked.

When I saw the child, on April 1st, he had much the general appearance of a case of nephritis; he was edematous over the whole skin surface, the face was extremely puffy, and the color presented a waxy pallor. He had at that time an effusion in the left chest that reached to about the third rib. There was no evidence of fluid in the right pleura, but there was considerable free fluid in the abdominal cavity. He was weak, at times, indeed,

extremely prostrated. There were numerous enlarged glands in both sides of the neck, in the axilla, and in the groins, the largest, on the left side of the neck, reaching the dimensions of a good sized hen's egg. As a result, probably, of previous X-ray treatment, the glands in the neck had become denser in consistency than is usually the case in Hodgkin's disease, and they had become somewhat adherent to each other. The cardiac impulse was displaced to the right, the apex beat being just to the left of the sternum. No evidence of enlarged mediastinal glands could be elicited. The fluid in the abdominal cavity made it impossible to determine definitely anything about the abdominal contents, excepting that the liver reached two fingers' breadths below the costal margin.

Upon inquiry Dr. Coplin informed us that examination of the glands removed at the Jefferson Hospital in July, 1904, had shown the lesions of early Hodgkin's disease. The boy was, therefore, spared the removal of other glands for diagnostic purposes.

On April 8th the fluid in the left pleura nearly filled this cavity and was causing distress. It was again removed, the amount aspirated being 1,500 c.c. It had the same appearance as that previously obtained. After standing for a day or two a jellylike coagulum formed in it, but the remaining fluid was still milky. The specific gravity was 1.009, the reaction faintly alkaline. There was a marked reaction for albumin; no reaction for sugar. Microscopically it had the same appearance as the other fluid. Smears and cultures showed no bacteria. The leukocyte count was then 15,400, and again nearly all leukocytes were polymorphonuclear. The examination of the urine again showed nothing but a slight trace of albumin with a very few hyaline casts.

The patient rapidly grew worse from this time on, had troublesome cough, marked dyspnea, and increasing prostration. His left pleura rapidly filled again, his general edema increased markedly, and he grew extremely weak. He died of exhaustion on May 10th.

The behavior of his temperature while he was under observation is of interest. When admitted he had constant fever, averaging about 101° F. This gradually decreased from March 5th, became normal on March 13th, and remained about normal for a few days; but it suddenly rose again on the 19th to over 103° F., and continued well elevated until March 31st, after this running

irregularly between normal and 101° F., until April 18th, when it again rose to over 103° F., and remained high until May 2d; it then decreased slowly and reached normal before his death, dropping indeed to 96° F. just before the end.

The autopsy was made by Dr. W. E. Robertson, whose report follows:

Body of a markedly emaciated boy. Lower extremities everywhere edematous. A long scar from the posterior auricular to the left clavicular region, marking the site of the early operation.

Thoracic Cavity.—The left pleural cavity contains a large amount of milky fluid. The left lung is compressed along the paravertebral groove. Heart and mediastinal contents pushed to the right. The right pleural cavity contains no excess of fluid. There are no adhesions, except between the lobes of the lungs. The left lung is collapsed and airless, and does not crepitate. It shows no scars. The right lung shows compensatory emphysema and is congested posteriorly. There are no scars. The lung crepitates throughout.

Pericardial fluid increased; fluid clear. There are two recent milk spots over the left ventricle. The valves are normal. Around the arch of the aorta, external to the posterior mediastinum, is an enormous mass of glands, which communicates with the chain of glands extending to the left axilla and to the neck. In the left clavicular region is a mass of glands and scar tissue, the whole mass being densely adherent to the operative scar.

Abdominal Cavity.—This contains a large quantity of clear serum, and there are several peritoneal cysts.

Liver.—Enlarged, displaced downward, and of nutmeg appearance on section; shows slight parenchymatous degeneration. Over the surface are numerous striæ, which appear to be dilated lymph radicles. Gall Bladder.—No calculi. The bile is thin and pale.

Spleen.—Fully twice the normal size. On section, shows numerous foci, which have a somewhat purulent appearance, but they contain no fluid pus.

Kidneys.—Of normal size. Capsule strips readily. The cortex is pale and of normal width; the pyramids are somewhat distended, suggesting interstitial change. Adrenals.—Enlarged, the left being flattened by an enormous retroperitoneal mass.

Pancreas.—Shows no gross change.

Mesenteric Glands.—Everywhere greatly enlarged and scat-

tered in chains along the mesenteric border. Retroperitoneal Glands.—From the inguinal region to the diaphragm, they are greatly enlarged; and they protrude through the diaphragm, so as to form practically a continuous mass with the posterior mediastinal glands. The iliac artery and vein, on both sides, are much compressed; and this probably produced the edema of the legs.

Microscopical Examination.—Cervical Lymph Glands.—All semblance of normal gland tissue is lost. There are very vascular lymphoid deposits about the vessels, and the vessel walls are thickened. There are numerous eosinophiles, which are especially polymorphonuclear. Many mononuclears are also found in the lymphoid aggregations. The lymphoid foci are widely separated by a stroma, which is, in part, actively proliferating connective tissue and also fibrin that has apparently come from hemorrhagic extravasation; and, in part, a structureless interstitial substance. Through this stroma are scattered erythrocytes, connective tissue nuclei epithelioid cells, lymphoid cells, and, in especially stained sections, plasma cells. Scattered throughout are many giant cells, some of them with three nuclei, and others with from eight to ten, the nuclei being in heaps or about the circumference. The capsule of the gland is not materially thickened.

Retroperitoneal Lymph Gland.—This is composed of structures similar to those already described, but contains only an occasional eosinophile and much less interstitial substance, the remainder of the gland being composed of blood lymph sinuses, containing numerous lymph corpuscles, the latter being vacuolated. The appearance of the gland is somewhat that of an atypical hemolymph gland. Van Giesen's stain shows considerable fibrous tissue, especially about the blood vessels, and in scattered foci everywhere. The great bulk, however, presents a fibrous appearance and takes especially a picric acid stain of a dark, yellowish brown tint in some places; in others, being decidedly yellow, indicative of its probable recent hematogenous origin.

Spleen.—Much congested, with hemorrhagic foci. There are a few eosinophiles. The vessel walls are thickened, especially the muscular coat, and show hyaline degeneration. A great many giant cells are present, particularly small ones, with four or five heaped nuclei. There are early degenerative foci, showing nuclear fragmentation, which is most marked in the stroma. The capsule is thickened, and fibrous bands are not unduly multiplied.

Liver.—There is some interstitial hepatitis, recent and old

and some multiplication of the bile ducts. The parenchyma stains poorly, the cells and nuclei being pale. The intercellular spaces are widened, and are often choked with erythrocytes; and there is widespread, almost uniform congestion, with scattered foci of hemorrhagic extravasation. There are no eosinophiles; but a few giant cells are found, always within or about the numerous collections of cells that are seen throughout the section. These cell collections are composed, in addition to giant cells, of aggregations of lymphoid polymorphonuclear and epithelioid cells. There is a very slight degree of fatty infiltration.

I secured three glands, one of them small, one medium sized, the third one of the largest; and Dr. Joseph Evans kindly injected these into the peritoneal cavities of six guinea-pigs at the William Pepper Laboratory of Clinical Medicine, portions of each gland being injected into two guinea-pigs. One of each of the three sets of animals was killed on June 15th; all had been entirely normal, and the autopsy showed nothing abnormal in any, with one exception. This animal had on the surface of its spleen two small bodies about one-eighth of an inch in diameter, of rounded surface and of much the same color as the spleen itself, though somewhat more grayish. They were apparently entirely separate from the spleen, and could be moved over the surface slightly, though attached by connective tissue. I examined sections of these, which Dr. Thomas Cope kindly made for me, and found nothing but lymphoid tissue with irregularly distributed, somewhat dense, fibrous tissue. There was no suggestion of tuberculosis, and the objects were apparently fibroid supernumerary spleens. The other set of guinea-pigs are still living and well.

The case, therefore, is a further contribution to the view that Hodgkin's disease does not have any essential relation to tuberculosis. This point seems to have been quite thoroughly established by a large series of observations in the past few years. The work of Dorothy Reed, followed by that of Longcope and others, seems to have shown that the presence of tuberculosis in cases of Hodgkin's disease is accidental, and is due merely to the ubiquity of tuberculosis, not to any primary relation to the glandular enlargement in Hodgkin's disease.

The case also adds evidence of the apparent value of an examination of the glands early in the disease in determining that Hodgkin's disease is present; the diagnosis made early in this

case at the Jefferson Hospital, as a result of microscopical examination of the glands, was confirmed by the later course of the case and by the autopsy. Basing a diagnosis upon microscopical examination of the glands postulates a belief that the morphological changes found in the glands in Hodgkin's disease constitute a pathological entity. This, I believe, has been demonstrated with the greatest probability. If large numbers of observations establish the value of this means of diagnosis with final definiteness, and it now appears very probable that this will be the case, it will be of the utmost value in that it will render a diagnosis that is otherwise often extremely difficult and at times almost impossible, a comparatively simple matter. We are already apparently justified in depending upon this method of diagnosis.

The differential count of the leukocytes needs but passing comment, though it is of interest because of the claim of Pinkus and others that the differential count in Hodgkin's disease shows a high relative proportion of lymphocytes. This view has, indeed, become rather general. The relative proportion of the lymphocytes is frequently high, but Longcope and some other observers have dwelt upon the fact that this is by no means always the case, and is not to be depended upon at all in diagnosis. This case is further evidence of the truth of the latter contention, the polymorphonuclears having been counted at over 95 per cent. of the total, and having at each inspection evidently constituted more than the normal proportion of the white cells, while the lymphocytes were correspondingly low.

The pleural effusion constituted, however, as I have stated, the most interesting and unusual point in the case. As I have already noted, some degree of milkiness of effusions due to substances other than fat is probably not uncommon; and it has been repeatedly shown by accurate chemical studies that a decidedly milky appearance closely resembling that due to large amounts of chyle may be caused by substances other than fat. In this instance the main characters of the fluid were determined to be as follows: It did not change its appearance upon standing even after weeks had passed, excepting that within the first twenty-four hours a clear jellylike coagulum of large bulk formed. Filtering the fluid, even through several layers of Schleicher and Schüll's densest filter paper, caused no change in its appearance and the same was true of prolonged centrifugation at a high speed. Filtering through a Berkfeldt filter, however, almost en-

tirely cleared the fluid, only a slight turbidity remaining, and filtering through a Pasteur-Chamberland filter cleared it completely, the fluid then being water clear and pale yellow in color. On the exterior of either of the porcelain filters one found after the fluid had passed a slightly sticky substance which, when washed off in distilled water and shaken in water, produced a milkiness similar to that seen in the original fluid. This milkiness was, however, plainly due merely to suspension of particles in the water, for after standing for an hour or two the particles all settled toward the bottom and the upper portions of the water became quite clear. The specific gravity of the substance was, however, low, and the settling occurred very slowly. Shaking the original fluid with ether after adding potassium hydrate caused it to clear only very slightly; the ethereal solution afterward contained a faint trace of fat, but the amount was extremely small. Quantitative estimation of the total fat in the fluid showed only 0.2 per cent.

Microscopically the slight deposit found after centrifugation showed a considerable number of lymphocytes, very few polymorphonuclear cells, no red blood corpuscles, and no other formed elements. There was a moderate amount of formless *débris,* but the most striking feature was furnished by the numerous highly refractive minute globules. The original fluid before centrifugation, as well as the supernatant fluid after centrifugation, showed very great numbers of these refractive fine globules. The globules had much the appearance of those seen in a very fine fat emulsion, but they did not stain with osmic acid or Sudan III. Small portions of the *débris* stained with these fat reagents, but very few, if any, of the fine globules did so. That the milkiness of the fluid was due to some substance other than fat is evident, therefore, from the observations that I have thus far described.

Boiling without the addition of acid caused considerable coagulation, but the fluid remained slightly turbid; adding a drop of acetic acid and boiling further produced a marked coagulum, and the filtrate was then perfectly clear. The other usual tests for albumins, such as potassium ferrocyanide and acetic acid, were, of course, markedly positive. Saturation with magnesium sulphate produced a very dense precipitate of very fine character; prolonged centrifugation did not clear up the fluid after this, but filtration yielded a perfectly clear filtrate. Half saturation with ammonium sulphate yielded a perfectly clear filtrate. Boiling

this filtrate, with the addition of a slight amount of acetic acid, gave a further marked precipitate of albumin. Albumoses were absent.

Apparently, then, precipitation of the globulin fraction without precipitating all the albumin carried down the substance that produced the milkiness. From microscopical examination it appeared that the milky look was due to the presence of the fine globules previously described. Before filtering through a Berkfeldt or Pasteur-Chamberland filter these were, as stated, present in very large numbers. After filtering through porcelain, the fluid that was previously clear contained none of these fine globules, while that which was still slightly turbid contained a few. Furthermore, the effect of the addition of gelatin to the fluid that had passed through porcelain indicates that these globules were the cause of the milky appearance. Some years ago, when working on the supposed effect of gelatin upon coagulation of the blood, I observed that the addition of gelatin to blood serum resulted in the development of a marked turbidity; and upon microscopic examination of this turbid mixture I found a great number of very fine, highly refractive granules which had the same microscopic appearance as those seen in this milky effusion. Because of this previous observation I tried the effect of gelatin upon the fluid obtained by passing this effusion through a Berkfeldt filter. While, as was noted, previous to the addition of the gelatin this fluid was almost clear and contained very few of the refractive globules, soon after a considerable portion of 10 per cent. gelatin solution (about 1 part 10 per cent. gelatin solution to 3 parts filtered effusion) was added, the mixture became decidedly turbid, and upon microscopical examination it showed again numbers of very fine granules and globules which were highly refractive, and resembled those that had been present before the filtration of the effusion. This was not due to the precipitation of phosphates; the particles had no resemblance to amorphous or crystalline phosphates, and, furthermore, the addition of acetic acid did not clear up the mixture of filtrate and gelatin, it, on the contrary, made the mixture become decidedly more milky in appearance, and increased largely the number of granules seen under the microscope. The explanation of this effect of gelatin is very possibly to be found in the observations of Henri, Lalou, Mayer, and Stodel concerning the influence of various colloids upon each other. These investigators state that

colloids of the same electrical affinities tend to hold each other in solution, while those of the contrary electrical affinities tend to precipitate each other more or less completely. Among the colloids that travel toward the anode, they mention gelatin; among those that travel toward the cathode, they mention hemoglobin. I am not aware of any such observations on serum globulin; but since hemoglobin belongs to the class of the globulins, it is somewhat probable that hemoglobin and serum globulin behave in the same way. If this is the case, the observations of the authors just mentioned offer a physical explanation of the effect of gelatin upon blood serum and upon this pleural fluid. They also suggest that the occasional development of this milky appearance in fluids of the body may be due to the physical effect upon each other of any colloids that have opposite electrical affinities. This question concerning the cause of the milky appearance of these effusions will, however, be referred to again in discussing the main points that have been demonstrated by the literature relating to milky effusions of this class.

Most of the literature has recently been gone over in detail by Bernert, and I need merely repeat much of what he has written, adding a few details that have been reported since the time of his article, and correcting one or two points of some interest in which Bernert is apparently in error.

Bernert gives to Quincke the credit of first directing attention to the fact that milkiness of effusions may be due to changes in the protein, and not to fat emulsion. Quincke does seem to have been the first to direct attention to this point with decided emphasis, but in 1872 Stevenson described a case of this sort and dwelt upon its interest quite as clearly and in much the same terms as did Quincke in 1875. Quincke's more serious consideration of the matter appeared still later.

Since then there have been a number of more or less complete studies of such effusions. Michele and Mattirolo, Apert, Ascoli, and Bernert have in particular added largely to the knowledge of the subject, the last mentioned author having discussed 36 cases (according to his table) that he collected from the literature.

These effusions occur under varied circumstances and apparently have no characteristic relation to any primary disease. They have been seen repeatedly in tuberculosis, in sarcoma and carcinoma of various abdominal organs, in heart disease, in dis-

eases of the liver, in nephritis, and in a number of cases in which no definite diagnosis was reached. The degree of cloudiness or milkiness may vary from one extreme to the other. It is well known that effusions not infrequently have a somewhat turbid or faintly milky appearance when fat is absent. Beginning with such slight cloudiness there are increasingly marked degrees up to those cases in which the fluid has a characteristically milky look, the latter cases being comparatively rare. It has repeatedly been noted, as was true in my case, that the effusion in one serous cavity is milky, while the fluid in other cavities is clear; sometimes, however, milky effusions have been found in two or more cavities in the same case.

Clinical or anatomical evidence of the escape of chyle into the effusions is not found in these cases, and the effusions have not the chemical or physical characters of chylous or chyliform effusions. The most striking points that show that they are not ordinary chylous or chyliform effusions are, first, the fact that the amount of fat is usually very small, and there may be practically none present; second, whatever the amount of fat found, removal of it does not do away with the milky appearance; and, finally, in every instance properly studied it has been determined that protein substances were responsible for the milkiness. The special protein substance causing the milkiness is always or nearly always probably of the class of globulins, and most frequently it is in all probability serum globulin.

It is of considerable importance to recognize the fact that fat may be present in milky effusions, and yet not cause any of the milkiness, or may produce at most only part of it. In Saracen's case, for example, the fat amounted to just 1 per cent., but his description makes it highly probable, though not certain, that much of the milkiness in this instance was due to protein. Sorgente, whose original article I could not obtain, is referred to by Bernert as having found over 0.6 per cent. of fat in his effusion, while it was determined that much of the milkiness was due to protein. Shaw reported over 0.5 per cent. in his case, and yet his description shows that the milkiness was little, if at all, due to fat; shaking with ether did not clear it in the least. There is a general tendency to attribute all milkiness to fat, if fat can be found even in small amount, and considerable stress has erroneously been laid by Bernert and a number of other writers upon the supposed observation of Letulle, that as little as 0.15 per cent. of fat is suffi-

cient to cause milkiness of the effusion. As a matter of fact the point that Letulle established was merely that his effusion was milky and contained 0.15 per cent. of fat. He did not show clearly that the milkiness resulted from this quantity of fat, though at that time he believed that he had shown this; on the contrary, careful reading of Letulle's report indicates pretty clearly that he unknowingly showed actually that the milkiness was not due to the fat. Whatever the amount of fat necessary to produce milkiness in a fluid, it is certain that the milkiness depends largely upon the physical condition of the fat, and not merely upon the amount present, for Bönninger finds 0.85 per cent. of fat the usual amount in normal blood serum, and as much as 1.4 per cent. in diseases. Much smaller quantities than this are known to produce milkiness, though these amounts did not. The determination that milkiness is dependent on protein and not upon fat depends upon the demonstration that removal of the fat either removes only a part of the milkiness or does not alter it. That is, in those cases that are recognized as being in the class under discussion, shaking with ether and an alkali leaves the fluid still milky. It has been repeatedly noted that extracting with ether may cause the fluid to become somewhat less milky, though it does not clear entirely; in such instances the lactescent appearance is evidently due partly to fat and partly to altered protein.

Beyond the fact that the amount of fat present is small, the main characteristics of the effusions are as follows: The specific gravity is usually rather low, and in most cases has been below 1.015. It has, however, been recorded as high as 1.082 (Apert). The reaction has nearly always been found slightly alkaline, but it has been found neutral and even slightly acid. Little, if any, odor is present. On standing there may be no change in the appearance of the fluid, or a very slight creamlike layer may rise to the top; examination of this layer may show that it is due to the presence of a small amount of fat, but it may also be due to protein substances and may contain little or no fat.

One of the most remarkable characteristics of these effusions is their resistance to putrefaction. This point has been repeatedly emphasized and is very striking. Several authors have observed that the fluid may be allowed to stand for weeks without showing any signs of putrefactive changes; and the effusion in my case, which was removed on April 10th, showed absolutely no change in character on June 5th. On June 10th, I found it decidedly

changed in appearance, not having observed it meanwhile; it then consisted of a clear yellowish fluid containing a fine coagulum, but there was still no odor of putrefaction. During these two months the fluid had been kept at night in the ice chest, but during the day it had many times stood for hours in an open vessel in a warm room, and there had been at no time any attempt to keep it from infection.

The most painstaking filtration of these effusions through paper has no effect upon their appearance, and the same is true of prolonged centrifugation. I added talc, calcium carbonate, and other neutral fine powders to the fluid, and after thoroughly stirring the filtrate centrifugated it for a long time and also filtered. This, too, was without effect, though it will usually clear up a turbidity in urine or other fluids that does not disappear upon filtration or centrifugation. Microscopical examination shows varying numbers of cells whose character depends to some extent upon the character of the primary condition producing the effusion; but the most striking microscopical appearance is the presence of very great numbers of small refractive bodies. These closely resemble a fine fat emulsion, because of their globular form and their very marked refractive power; so closely indeed that their presence as well as their absence has led a number of authors into very positive statements concerning the presence or absence of fat. They may, however, be shown to be not fat by shaking with ether and an alkali and observing that they persist, or by staining with osmic acid or Sudan III, and noting that they do not take these fat stains. The fat dyes may stain a more or less considerable number of particles, depending upon the amount of fat in the effusion, but the important fact is that the majority of these small globules do not take such stains. These fine globules or granules have, also, in most instances, been observed to have a decided molecular motion. They are of the same appearance as those that have been seen at times in milky looking blood serum, and they are, as I noted, of the same appearance as those that I observed after adding gelatin to blood serum. Apert and Bernert passed the effusions they studied through porcelain filters, and I did the same. In all instances the milkiness was completely or almost completely removed by this procedure. I examined the effusion in my case after it had passed the porcelain filter, and observed, then, that the fine particles had disappeared coincidently with the disappearance of the milkiness.

Apparently, then, these particles give rise to the milky appearance.

The effusions, of course, give the usual reactions for albumins, and fairly complete precipitation by any method of all the protein present removes the milkiness. The milkiness appears, however, in all instances to be produced by some substance that is separated out with the globulins. Ascoli, by prolonged dialysis, caused the fluid to clear up markedly, and at the same time there was separated out a globulin-like precipitate; and all those that have studied this point have found that precipitating out the globulins by means of half saturation with ammonium sulphate or saturation with magnesium sulphate causes the fluid to become entirely clear.

There has been considerable discussion as to the exact cause of the milkiness. A number of authors, Michele and Mattirolo, and Mosse in particular, have claimed that the milkiness was due directly to lecithin; it is known that this substance may cause turbidity of fluids when present in considerable amount. Bernert's study of the literature, however, and the recent observations of Christen have shown that the amount of lecithin in both milky and non-milky effusions varies greatly, and shows no definite relation to the physical appearance of the fluid; and, furthermore, substances that will dissolve lecithin will not clear up the milkiness. As has been noted, the milkiness apparently stands in a close relation to the globulins, since separating out the globulins always clears up the fluid, but it is certainly not directly due to the presence of a large amount of globulin, for, as was just noted in regard to lecithin, the amount of globulin has varied greatly and has shown no relation to the presence or absence of milkiness. Bernert believes that the milky appearance is due to a combination of lecithin with globulin, which alters the character of the globulin and gives rise to a partial precipitation of the latter. In support of this view he notes the fact that the addition of lecithin to solutions of some proteins is followed by precipitation or partial precipitation of the protein as a result of combinaton of the lecithin with the protein; and he also determined in his cases that lecithin in considerable amounts was found in the globulin fraction, apparently combined with the globulin. It seems to me to be probable that the cause of the milkiness varies in different cases, and that it may be due to the presence of various substances which have the power of causing alterations in the globulins which lead to a partial precipitation. I have referred to my ob-

servation concerning the influence of gelatin upon blood serum, and its influence upon the effusion in my case after the fluid had been cleared by filtering through porcelain. This indicates that in my case, at least, an identical or very similar change in the fluid was produced merely by the presence of another protein. This action of gelatin is, of course, probably due to the differences between its physical characters and those of globulin; perhaps the specific difference that determines the change is the behavior to electricity. At any rate, the effect of gelatin strongly suggests that a number of substances that might escape into effusions may be capable of producing this effect, and it is probable that no one substance is always responsible. The main point of interest at present is, however, the fact that the milkiness is due merely to a change in some protein present in the effusion, not to chylous or other fat; and this change is apparently a partial precipitation or emulsification of the protein, the change not being sufficient to cause it to separate out completely in the form of a definite precipitate. The condition is similar to that which has in a few instances been observed in the blood serum and in the urine. Widal and Sicard have discussed non-fatty lactescence of the blood serum and the literature relating to it, while Cramer and Coriat have described cases in which the urine had a milky appearance which was not due to fat, but to albumin which was present in the form of fine globules, similar to those that are seen in these effusions. The milkiness is also directly comparable to the appearance of actual milk from which the fat has been removed. The studies of Picton and Linser and others have demonstrated that the albumins in milk are present in various forms, the lactalbumin in actual solution, the casein in suspension or partial precipitation. The latter gives rise to the bluish white opaque appearance of the fluid.

In conclusion, then, I would say that a milky appearance of animal fluids may be due to three causes. The two that are most commonly mentioned are the presence of actual chyle, this constituting a chylous effusion, or the presence of degeneration fat without any actual escape of chyle into the effusion, this constituting a chyliform effusion. The third cause is the presence of altered protein, probably usually altered globulin, the milkiness in this instance being only partially, if at all, the result of the presence of fat. The only way of determining whether the milkiness is due to fat or protein is not merely to estimate the amount

of fat present, but to determine whether removal of the fat with ether and an alkali causes the milkiness to disappear. If it does, the milkiness is due to fat, but it has not yet been determined that the milkiness of the effusion is definitely due to admixture of chyle and not to degeneration fat. It becomes more probable that it is chylous if substances that are easily recognizable by their color are ingested, and are a short time afterward found in the fluid removed by puncture; and the probability that the fluid is chylous may also be increased by study of the chemical character of the fat, for chyle fat and degeneration fat apparently show chemical differences. The latter point, however, is not definitely established, and it is often very difficult, particularly without post-mortem examination, to be entirely sure that a fatty effusion is chylous, and not chyliform. If shaking the fluid with ether and an alkali leaves it still milky, the appearance is due to altered protein. It is probable that in a fair proportion of cases of milky effusions proper study will show that the lactescence is due in part to altered proteid, even though a good deal of it may be determined to be due to fat.

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DISCUSSION.

DR. GRAHAM.—There are one or two points in the paper I should like to speak about. First, in regard to diagnosis, I think a great many cases have been described as Hodgkin's disease that really do not properly belong under that term. Last winter I had a case under observation where before death certain glands were removed and at autopsy portions of various organs were examined. The most careful search failed to show anything of a tuberculous nature. I think it is rather important to remove glands where it is possible in these cases before death, so that a microscopical examination may be made when removed from the living rather than the dead subject. That throws out a possible error arising from changes that take place after death. I think it will help to corroborate the work Dr. Edsall speaks of, that Hodgkin's disease is an entity and can be proven so microscopically.

DR. EDSALL.—I should like to ask the members what experience any of them may have had with the use of X-rays in Hodgkin's disease. I have seen 2 or 3 cases in children in which there was a temporary, almost complete, disappearance of the glands, but most of the cases have relapsed.

DR. JACOBI.—I have observed 1 case for quite a number of months and at first there was a great reduction in size of the glands and there appeared to be a general improvement but it only lasted six or eight weeks and then there was a relapse.

DR. HUBER.—I believe that is the same case we had in the hospital and where an extensive enucleation of the glands was made afterwards. The child was better for a while, but soon relapsed and recently died, about a year and a half after it first came under observation.

DR. TOWNSEND.—I have seen 1 case that improved very much for a short time, but soon the disease increased and the child died. There was, however, some doubt as to whether this was a case of tuberculous glands or of Hodgkin's disease.

AN ANALYSIS OF 145 CASES OF TYPHOID FEVER IN CHILDREN.

BY ALFRED HAND, JR., M.D., AND J. CLAXTON GITTINGS, M.D.,
Philadelphia.

The utility probably exists of analyzing the records of a series of cases, although nothing of special interest or novelty may appear, even when the disease under consideration is such a common one as typhoid fever. We often hear the remark made by the Nestors of the profession that such and such a disease is not what it used to be, and although it is a well-known fact that a disease varies in severity in different epidemics, yet the general trend of disease can only be estimated by careful study of groups of cases. Such studies may have some value if the treatment has been as much as possible consistently the same. It is also not beyond the range of reasonable hope that preventive medicine will at some time be so widely recognized throughout our whole land that the disease under consideration, as well as some others, may be conspicuous by its absence. If that consummation devoutly to be wished for ever arrives, case-histories and analyses of cases may come to possess historical value. Such are our reasons for presenting the dry bones of figures of 145 cases of typhoid fever seen by one of us in the Children's Hospital of Philadelphia. Acknowledgment is due to Dr. Alfred Stengel and to the memory of our lamented fellow-member, Dr. Frederic Packard, for cases seen while substituting in their services in 1902, the remainder having occurred in the service of one of us during 1903 and 1904, from April to July, inclusive.

Age.—The accompanying diagram shows the age-incidence graphically, two points in connection with it being worthy of comment. One is the number of infants affected, ten, which is an unusually high percentage in our own experience. The diagnosis was confirmed in all of these cases by a positive Widal reaction. The chart also seems to bear out an inference drawn in an earlier series of cases (Hand and Walker, *American Journal of the Medical Sciences,* June, 1902) that the period of the second dentition, with its accompanying disturbances of digestion, is an important predisposing cause of typhoid fever. Thus, considerably

over one-third (37 per cent.) of the cases fell in the fifth, sixth and seventh years.

Sex and Race.—Fifty-seven per cent. were boys, 43 per cent. were girls, 85 per cent. were white, and 15 per cent. were colored. These figures show no decided variation from those for the general population of the hospital.

Prodromal Symptoms.—In attempting to estimate the value of the histories prior to admission, certain limitations are to be recognized, chief of which are the intelligence and powers of observation of the parents or guardians, and, perhaps to a lesser degree, the personal equation of the admitting officer; in some cases, most diligent questioning can elicit nothing of value, and often the parents are of foreign birth and no richer in their knowledge of English than in the coin of the realm. In two patients nothing at all could be learned of the previous sickness, so that the following statistics refer to 143 cases.

Fever	was	present	in	100	per	cent.
Anorexia	"	"	"	78	"	"
Headache	"	"	"	62	"	"
Diarrhea	"	"	"	55	"	"
Abdominal pain	"	"	"	53	"	"
Drowsiness	"	"	"	46	"	"
Vomiting	"	"	"	42	"	"
Constipation	"	"	"	35	"	"
Nosebleed	"	"	"	25	"	"
Delirium	"	"	"	16	"	"
Chill or chilliness	"	"	"	10	"	"

Sore throat	was	present	in	1.4	per	cent.
Pain in chest	"	"	"	1.4	"	"
Vertigo	"	"	"	1.4	"	"
Nausea	"	"	"	1.4	"	"
Vomiting of blood	"	"	"	0.7	"	"
Convulsions	"	"	"	0.7	"	"

Day of Disease on Admission.—This was more difficult to determine than the prodromal symptoms, but in the majority of cases the statements are fairly accurate, and the admissions were:

3	on	the	first	day
2	"	"	second	"
6	"	"	third	"
12	"	"	fourth	"
19	"	"	fifth	"
21	"	"	sixth	"
23	"	"	seventh	"
15	"	"	eighth	"
7	"	"	ninth	"
16	"	"	tenth	"
3	"	"	eleventh	"
3	"	"	twelfth	"
1	"	"	thirteenth	"
7	"	"	fourteenth	"
3	"	"	fifteenth	"
1	"	"	sixteenth	"
1	"	"	twenty-first	"

After admission to the hospital, the maximum temperatures were as follows:

From	101°	to	102°	in	3	cases, or	2	per	cent.
"	102°	"	103°	"	8	" "	5.5	"	"
"	103°	"	104°	"	21	" "	14	"	"
"	104°	"	105°	"	52	" "	35	"	"
"	105°	"	106°	"	49	" "	33	"	"
"	106°	"	107°	"	11	" "	7.5	"	"
"	107°	"	108°	"	1	" "	0.7	"	"

The severity of the disease can easily be inferred when it is seen that over three-fourths of the cases developed fever of 104° or over.

The shortest course which the fever ran was eight days in 1 case, while the longest was forty-six days. The average duration of the febrile course in the cases going on to recovery was twenty-five days, the uncomplicated cases giving an average of twenty-one days and those having complications running an average of twenty-six days.

The shortest stay in the recovered cases was fifteen days, while the longest, due to a relapse and subsequent furuncles, was seventy-seven. The average number of days in the hospital was thirty-one, and this happens to be the same for the recovered cases and for all the cases, including those that died or were removed before recovery.

In the hospital the ordinary symptoms of typhoid fever were developed in the following order of frequency:

1. *Widal reaction.* This was positive in 138 of the patients, or 95 per cent. The first test made gave a positive result in:

1	case	on	the	first	day
1	"	"	"	third	"
4	"	"	"	fourth	"
10	"	"	"	fifth	"
18	"	"	"	sixth	"
14	"	"	"	seventh	"
13	"	"	"	eighth	"
17	"	"	"	ninth	"
12	"	"	"	tenth	"
10	"	"	"	eleventh	"
5	"	"	"	twelfth	"
4	"	"	"	thirteenth	"
3	"	"	"	fourteenth	"
5	"	"	"	fifteenth	"
3	"	"	"	sixteenth	"
2	"	"	"	eighteenth	"
1	"	"	"	twenty-third	"
1	"	"	"	twenty-fourth	"
1	"	"	"	fortieth	"

In twelve patients the reaction was negative at first, but subsequently became positive, as the following table shows:

Negative	on	the	fourth	day,	positive	on	the	eighth	day
"	"	"	eighth	"	"	"	"	tenth	"
"	"	"	third	"	"	"	"	eleventh	"
"	"	"	seventh	"	"	"	"	twelfth	"
"	"	"	eighth	"	"	"	"	"	"
"	"	"	"	"	"	"	"	thirteenth	"
"	"	"	ninth	"	"	"	"	"	"
"	"	"	eleventh	"	"	"	"	fourteenth	"
"	"	"	fifth	"	"	"	"	sixteenth	"
"	"	"	twelfth	"	"	"	"	"	"
"	"	"	fifteenth	"	"	"	"	seventeenth	"
"	"	"	eleventh and fifteenth	"	"	"	"	twentieth	"

In 7 cases no positive reaction was obtained, but not much value is to be attached to this, when the attendant circumstances are considered. Thus, the test was negative on the seventh day, and before another test was made diphtheria developed, and the child was removed from the hospital; it was negative on the tenth day, when the child was admitted, but death occurred three days later and an autopsy made the diagnosis; in 2 other cases but one test each was made, on the eleventh and on the sixteenth days; in another case two tests were negative, on the ninth and thirteenth days; three tests were negative on the sixth, ninth and twentieth days in 1 case, and on the eighth, sixteenth and twenty-first days in another.

2. *Coating of the tongue* was present in 129 patients, or 80 per cent.

3. *Spots* were present in 87 patients, 70 per cent. of the white children.

4. *Enlargement of the spleen* was found by palpation in 95 patients, and in three there was great tenderness, giving 69 per cent. It was not palpable at any time in 26 patients, and in 21 its condition was not recorded.

5. *Abdominal tenderness* or *pain* was noted in 54 patients, or 37 per cent.; and *abdominal distension* was found 40 times, or 20 per cent.

6. *Condition of the bowels.* Thirty-three, or 22 per cent., were constipated throughout the attack, and 78, or 53 per cent. became so after a diarrhea of more or less severity; thus 57 had from one to four stools daily for a time and 21 had more than four stools daily, all becoming constipated later. Only 35, or 24 per cent., did not become constipated, and of these, 3 had natural movements throughout the course of the disease, 12 had a maximum of 4 stools daily, 14 had a maximum of 8 and 6 had a maximum of 13.

7. *Condition of the nervous system.* Delirium was present in 26, or 18 per cent.; 13 were drowsy, 11 were irritable, 2 were restless, 2 had convulsions, and 1 each had stupor and delirium; delirium and external strabismus; stupor and retraction of head; rigidity of neck and limbs; retraction of head; apathy.

8. *Condition of the lungs.* Thirty-eight, or 26 per cent., had bronchitis, 7 had bronchopneumonia, 6 had croupous pneumonia, and 1 pleuropneumonia.

9. *Leukocytes.* Blood-counts were made 121 times, 103 being in uncomplicated cases. In but 9 cases was there a leukopenia of less than 5,000 leukocytes per c. mm. Seventy-one gave counts of from 5,000 to 10,000, while 31 uncomplicated cases had from 10,000 to 16,000 leukocytes. Eighteen complicated cases were equally divided, half having a leukocytosis, of 11,000, 16,000 (3), 17,000 (3), 23,000 and 43,000; the other half ranging from 5,000 to 10,000. As a rule, however, the leukocyte-count furnishes valuable aid in the diagnosis, a normal count indicating typhoid fever, but a moderate leukocytosis not excluding it.

Complications were present in 45, or 31 per cent., in varying combinations.

Diphtheria	occurred	in	13	cases
Pneumonia	"	"	13	"
Otitis	"	"	13	"
Furunculosis	"	"	12	"
Hemorrhage	"	"	8	"
Measles	"	"	2	"
Bedsore	"	"	1	case
Gangrene of the skin	"	"	1	"
Perforation	"	"	1	"
Pleuropneumonia	"	"	1	"

Scarlet fever	occurred	in	1	case
Nephritis and convulsions	"	"	1	"
Ischiorectal abscess	"	"	1	"
Convulsions	"	"	1	"

Of the 13 cases of diphtheria, 9 were removed from the hospital immediately, 2 died in the hospital, and 2 recovered. One of the last group had had pneumonia earlier in the course of the typhoid, and then developed an extremely toxic form of tonsillar and faucial diphtheria, of explosive onset, with extensive formation of false membrane; the condition was so critical that removal from the hospital seemed out of the question, so the patient was isolated and large doses of antitoxin (4,000 units every six hours) were given for several days, until a total of nearly 70,000 units had been given, with steady improvement and perfect recovery.

Relapses occurred in seven patients, a percentage of 4.8 per cent. Death occurred in one, on the twentieth day, the relapse having begun on the thirty-second day of the disease after an apyretic interval of six days. In the other cases the onsets were on the sixteenth, nineteenth, twenty-first (2), twenty-fourth and forty-second days, the fever lasting respectively eleven, twenty-seven, nine, ten, fourteen and sixteen days.

The deaths numbered thirteen, or 8 per cent., of which two were uncomplicated. The complications and maximum temperatures in the other fatal cases were as follows:—

CASE I. Furunculosis, cellulitis of scalp, otitis media, convulsions, 105.4°.

CASE II. Diphtheria, 106°.

CASE III. Slight hemorrhages, bed-sore, bronchopneumonia, nasal and oral diphtheria, 105.4°.

CASE IV. Croupous pneumonia, gangrene of the skin, 107°.

CASE V. Bronchopneumonia, 105.8°.

CASE VI. Bronchopneumonia, nephritis, perforation and peritonitis, 105.2°.

CASE VII. Slight hemorrhages, double otitis, pneumonia and pleural effusion, 106.4°.

CASE VIII. Bronchopneumonia, 106°.

CASE IX. Slight hemorrhage, lobar pneumonia, 106.3°.

CASE X. Croupous pneumonia and convulsions, 105.6°.

CASE XI. Croupous pneumonia, 105.4°.

For the treatment of typhoid fever we are believers in the advisability of rest in bed and a liquid diet, coupled with hydrotherapy, if the pulse and temperature call for it, and the use of drugs as they seem to be indicated. With regard to hydrotherapy, 26 patients did not develop sufficient fever to need more than the routine daily cleansing bath. In 5 patients, bathing was done when the fever rose to 102.5°, but in the remainder 103° was used as the guiding point. Tub baths were given to 117 patients, the great majority being kept in the tub for five minutes in water of 85°F.; in a moderate number, depending on the reaction of the child, modification was made to 80°, 90°, 95° and 100°. Ten patients stood the tubs poorly and resort was then had to sponging, ice rub, or cold pack, with a mustard tub in one instance. Sponges were used in 24 patients, occasionally substituting the tubs, but more frequently the sponges failed to produce the desired benefit and the tubs were then given. In a few charts it was noticed that the most decided results from the tubs on both the pulse and temperature were obtained at the same hours on a number of successive days and the thought arose that perhaps certain nurses were able to handle the patients in such a way as to produce a more decided action; conversation with several nurses confirmed this idea, which seems reasonable, as children are human beings and have their likes and dislikes; this feature, however, was noticed, as stated, in only a few instances.

In the former series of cases analyzed we found that we had used whiskey nearly as a routine measure, but in the present series the aim was adopted to use it only when the pulse seemed to indicate it. It was always the first stimulant used, but it was found possible to conduct 52 patients to recovery without a dose of whiskey. The initial dose was governed largely by the age of the patient, 5 minims for infants, 20 minims for children under four years, ½ dram for older children, usually given in connection with the bathing, and the amount raised or lowered in accordance with the effect produced. Ninety-three patients received whiskey and 44 had also the next stimulant on the list, strychnin. Digitalis was given to 26 patients, mainly in emergency by the resident physician, as we (Hand) do not believe that its use is of help more than only occasionally; two patients seemed to be distinctly benefited by it, but the rest seemed unaffected. To 5 of the patients with pneumonia, nitroglycerin was given in emergency as a stimulant, which does not seem to

us to be rational. For diarrhea, bismuth salicylate was used in 19 cases, the subnitrate in 6 and betanaphtol bismuth in 1, while opium and silver were used in 7. With the exception of nux to 35 and cascara to 33 in convalescence, the rest of the medication may be termed "scattering," one or two receiving asafetida, turpentine, etc.

With regard to the continuance of the liquid diet in convalescence, we have become a little more liberal than formerly and do not adhere to the ten-day rule so strictly. If the tongue becomes perfectly clean, if the spleen is no longer palpable and if the abdomen is not distended, then soft food is given a few days after the temperature has remained normal, but even so, the majority were not started with it until seven, eight or nine days after defervescence. In 4 cases we were compelled to wait until fourteen, fifteen or sixteen days before it was judged safe to give solid food.

DISCUSSION.

DR. BUCKINGHAM.—The writer gave 69 per cent. of cases of enlarged spleen. I know that at any age there may be typhoid without enlargement of the spleen, but I think the general statement as to the difference between enlargement of the spleens of children and adults is wrong. My attention was called to this some years ago by a paper with which I did not agree, although it was written in part from my cases. That led me to make a personal examination for some years of all the cases in my service. In those years I think there were no cases in which the spleen was not found to be enlarged at some stage. If careful examination be made it may be found that the enlargement varies; it may be found only once or twice a week in a given child.

DR. ADAMS.—I read before the Maryland Medical and Chirurgical Faculty two years ago a study of 300 cases of typhoid fever in children and it is rather singular that the conclusions reached by Dr. Hand conform so closely to those I came to. The ages in my series I think were a little older; the greatest number in my collection were about eight or nine years of age—that is, as I stated, about the beginning of school life. As to the sex, the cases were about equally divided but I drew a strong contrast in regard to color; there were many more white than colored children affected with typhoid. As to premonitory symptoms, in my experience they were very indefinite. As a rule, in the Children's Hospital we do not get the patients until they have been treated outside for some time, often without a definite diagnosis. The typical symptoms are usually developed pretty completely by the

time we receive the patient, for it is rare that we get them before the tenth day. I am inclined to agree with Dr. Hand that the spleen is not so uniformly enlarged as in the adult and when you have once found it I think you can generally find it afterwards. The spots were present in a large percentage of cases. The mortality was the same, 8 per cent. In the discussion that took place at Baltimore Dr. I. E. Atkinson said that he was surprised at that mortality, and that in Baltimore it was rare that a child died of the disease. Dr. Osler came to my rescue, however, by saying that I was talking of hospital cases and not private practice and that many of these hospital cases die within twenty-four hours after admission to the institution.

As to treatment, and particularly hydrotherapy, I will state that so far as abscesses, otitis media, etc., are concerned just as many cases occurred in the group that did not receive bathing as in those that did. I doubt whether the tubbing has anything to do with it and rather think it is the septic condition that produces the complications. In the cases where large doses of quinin were used and in the cases treated later by the antipyretics there was a higher mortality. While I have not anything particular to say against resident physicians as a class I think we do occasionally get hold of a fool, and such a man can do more harm in a day than the wisest physician can remedy in a long time. I remember one such resident physician who had read that guaiacol could be used in typhoid and he rubbed one of the babies with it until he produced an alarming condition. In the same way I have sometimes found them giving antipyretics and other remedies to my typhoid patients without my knowledge. The last period of treatment considered in my review was the diet and rest treatment and the best results were obtained in this group. I am surprised at the recommendation for the routine use of whiskey, for I consider that the vast majority of children with typhoid need very little stimulation.

Dr. Hand did not mention among the nervous symptoms cases of temporary insanity. I have reported to this Society 3 cases and Dr. Acker and I have since seen 2 more.

DR. GRIFFITH.—I would like to ask Dr. Hand whether he said that he had 1 or 2 cases of perforation and, if so, whether there were any symptoms different from those found in adults. I have recently reported 6 cases seen in my practice, and I believe that the accident offers difficulties still greater than it does in the adult patient.

There are two matters of which I want to speak pertaining to our practice in the Children's Hospital of Philadelphia. The first relates to the use of the tub bath. We never use the rigorous Brand treatment. The water is never over 85° at the start, though it sometimes cools down later. Many children will not, however, tolerate it as low as this, and even the actual warm bath

of 90 to 100° is sometimes not well borne. I am convinced that those of us who are teachers ought to impress upon our students the necessity of modifying the application of cold baths in the case of children. It is often capable of doing far more harm than good.

The second point, at least as regards my own custom, is that I have been abandoning the strict typhoid diet. It seems to me certain that those writers are correct who claim that the long continuance of debility, and the degree of emaciation often seen in typhoid fever depend upon the fact that not sufficient food has been given. We cannot obtain a sufficient number of calories from milk, unless we give a very great excess of it. Therefore, when I find a child crying for food, particularly as convalescence is approaching, I do not hesitate to increase the amount and the variety. One repeatedly encounters difficulty in cases seen in consultation, because the attending physician fears to give anything to eat as long as the slightest fever continues. It is not infrequent that the absence of food is really the cause of the persistence of fever.

DR. BLACKADER.—What do you give them?

DR. GRIFFITH.—The usual articles of "soft diet," such as cereal gruels, beef juice, milk toast and similar foods.

DR. BLACKADER.—I have listened to this paper with great interest. In regard to the enlargement of the spleen in typhoid fever in children I am quite confident that the enlargement cannot always be made out by palpation. Increased splenic percussion dullness is almost always present.

In reference to the Brand treatment of typhoid fever in children, I am inclined to agree with Dr. Griffith. Children do not stand prolonged or severe cold, as heat is abstracted more rapidly from the surface of the body in a child than in an adult, and their power of reaction is less. I think, however, that we obtain from tubbing, effects not obtained from sponging or any other of the milder applications of cold or cold water, but the temperature of the bath must not be too low and the duration of it not as prolonged as often employed; I rarely exceed four or five minutes. I fully agree with the remarks of one of the speakers as to the necessity for caution in the use of alcohol. There was a time when I thought its employment necessary in the majority of cases, but every year I am becoming more and more skeptical as to its value in this affection except as a stimulant to the digestion and absorption of food. In those cases in which nourishment is taken freely and the food is digested, an alcoholic stimulant is not called for. The advice of Murchison, given many years ago, was not to begin stimulants too early.

I cannot understand any conditions arising in the course of typhoid fever in children which would call for the employment of nitroglycerin. There is, I think, a good deal of misapprehension as to the exact action of nitroglycerin. Some authors speak of it as being a heart stimulant. It may be for a mere fraction of a minute, but if so the period of time is too short to be recognizable by instruments. It dilates the blood vessels paralyzing the muscles of the arterial walls and the only conditions under which it is of service to the circulation and the heart are those in which the resistance in the vessels is relatively increased and the blood pressure relatively high; conditions which seldom or never occur in typhoid fever in children.

DR. ABT.—Some years ago I experimented with the bath treatment of typhoid fever in children. I very soon came to the conclusion that cool baths were contraindicated in children. They dreaded each succeeding bath, and they suffered some shock. Warm baths, the temperature varying from 90° to 95° F., lowered the temperature and were not disagreeable to the patient; all in all they seemed to answer the purpose very well. After I had treated a considerable number of patients in this way, I had charts constructed which showed the amount of reduction of temperature during the course of the illness. The charts showed that the temperature fell two or three degrees and sometimes more with each warm bath. At times we observed that if the temperature had not fallen sufficiently it could be effectually reduced by leaving the child in the bath ten or twelve minutes instead of the usual five or eight minutes. This nearly always reduced the temperature the desired number of degrees.

DR. SAUNDERS.—In reference to the case Dr. Hand mentioned of dysentery giving the Widal reaction, we had a case some time ago of colon bacillus infection that gave a high reaction and made the diagnosis very puzzling for a few days.

As to hydrotherapy, I quite agree with the gentlemen who have spoken against the severe Brand method for children, and I would apply the same argument to many cases of typhoid in adults. I would also like to protest against the method of sponging in children as a necessary measure. Time and again I have seen the temperature rise during the treatment. The children fight against it and their muscular activity increases the body heat; worse than that, the skin is made a non-conductor. The spray bath I have found very good but it is not often available in private practice. It is the quickest way, and sometimes within one minute the temperature will fall two degrees using water of only 80° F.

I remember Murchison's advice, and he says that 6 ounces of alcohol a day is the limit to be given to adults. In children we

should be even more careful, and likewise with nux vomica or strychnia; digitalis is out of place when there is cardiac degeneration in any disease. We may use it in the beginning but not later in severe cases.

DR. HAND.—I would like to correct some misunderstanding of my paper. I agree with Dr. Blackader in regard to nitroglycerin, and want to say that I used it for lowering the blood pressure only. The other point was Dr. Adams' remark about the use of whiskey as a routine measure. It was only in the first series of cases that I found it was being used by the house physician and I kept it from the last 52 cases.

As to palpation of the spleen, I have examined for the past year the abdomens of all the children and found it visible some days below the costal border and not palpable on other days.

As to the question of perforation, one case came in just after perforation, which was not revealed until autopsy. At the same time I had another case which bothered me a great deal. It was treated symptomatically and got well. From my limited experience with perforation I would say that it is almost impossible to recognize it in children.

As to insanity, I should say that I excluded the mild deliriums and have never seen any case of real insanity following typhoid.

As to the diet, I had always aimed to keep the child on liquid until the fever had been gone a week or ten days, but I have come to modify that. With regard to the question of race, I found in the first series of cases that the proportion was the same as the proportion of colored children in the hospital from all causes.